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PROCTOLOGIC EXAMINATION AND DIAGNOSIS

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JUSTIFICATION for the choice of such an elementary subject for discussion in this paper may be found in the words of the late great surgeon, William J. Mayo, who wrote, "The progress of medicine has been so rapid that those conditions which are of major importance have occupied our attention and to a large extent have thrown out of perspective common ailments which may endanger life and always are a serious source of worry and annoyance to the patient. Especially is this true of diseases of the rectum and anus. Because of the nature of the complaint, the patient dislikes to speak of it, and too often he continues to suffer from hemorrhoids, fissure, fistula, and similar disorders which readily can be relieved.

"There is need for better work in proctology, by the surgeon, the internist, and the general practitioner. Of these three members of the profession, the general practitioner stands in the position to do the most good. The time when he first sees the patient is the most favorable time to avert lurking tragedy by accurate diagnosis and opportune treatment."

The wisdom of these words is borne out by the experience of several of the larger clinics of this country, where it has been found that the average time elapsing between the onset of symptoms and the establishment of the diagnosis of cancer of the rectum is nine months. There is no doubt that this delay is due, in large part, to the inherent disinclination of the average individual to seek medical advice for any condition until pain becomes a prominent symptom, and to a peculiar secretiveness about his condition when the difficulty involves the rectum. But, too often, that delay can be attributed solely to the physician. It is amazing that a physician should

find it unpleasant or undignified to examine a region of the body that is so important and so useful, upon which the life, health, comfort, and convenience of the individual is so dependent. It is unpardonable for a physician to be too busy to perform a rectal examination. When symptoms have become sufficiently important in the mind of the patient that he feels impelled to consult the doctor, it is imperative that the physician inquire into the nature of the disorder and make such examinations as are required in order to make the diagnosis. To prescribe empirically the convenient suppository or ointment is untenable. Used in such manner, they conceal more disorders than they cure. It has often been said that the lethal potentialities of the suppository are just as great as those of the death-dealing metallic missile whose shape it bears.

It has been estimated that one patient out of every seven is suffering from some disease, the relief of which would be assisted, or entirely accomplished, by the treatment of pathologic conditions discovered only upon rectal examination. How true, then, is the tenet that "No physical examination is complete without examination of the rectum!"

The diagnosis of anorectal diseases, like the diagnosis of disease in other regions of the body, is accomplished in three steps. An accurate history, followed by a careful examination, and supplemented by such laboratory examinations as may be indicated or necessary, should be the procedure in every case presenting rectal symptoms.

Chief Complaint: The first entry in the recorded history should be the chief complaint—a statement, in the patient's own words, giving the symptom which precipitated the visit to the doctor's office. The practice of allowing the patient to give his own diagnosis of the trouble should be avoided, especially in view of the tendency of the layman to refer to all rectal complaints as "piles." In eliciting the remainder of the history, the questions of the physician should be direct, systematic, and to the point, but not leading. Quite often, symptoms which may seem quite insignificant to the patient will have great bearing on the case.

Bleeding: The most common of all symptoms of rectal disease is bleeding. Bleeding from the rectum is never normal. The physician should determine the duration of the bleeding, and whether it is constant or intermittent; its relation to the bowel movement; whether it is bright red, dark red, or black; whether it merely streaks the stool, is mixed with the stool, or is profuse. The bleeding from an anal fissure or ulcer is usually slight in amount; that from internal hemorrhoids, various inflammatory lesions, prolapse,

polyposis, and malignancy is quite likely to be more profuse. In cancer of the rectum and in ulcerative coloproctitis the blood will usually be mixed with pus, mucus, and debris. It is a commonly accepted idea that bright red blood is fresh blood and comes from some pathologic process in the anal canal or rectum; that dark blood has remained in the bowel for a short time and probably is from the region of the sigmoid; and that black blood comes from a site higher in the intestinal tract such as the stomach, duodenum, or, perhaps, a Meckel's diverticulum, but this is not invariably true.

Pain: Bleeding from the rectum rarely causes a patient to seek medical advice until it has assumed alarming proportions. Human nature being what it is, however, pain will usually prompt the individual to seek relief at an early date. It is regrettable, then, that pain is a late symptom of cancer of the rectum and usually does not occur until the lesion infiltrates adjacent organs, becomes obstructive, or is situated so low in the bowel as to involve the anal canal and lie within the grasp of the sphincter muscle. The pain of which the rectal patient might complain varies widely along the full scale of disagreeable or painful sensations. The physician should determine its character, whether it is stinging or aching, sharp or dull, intermittent or constant. He should inquire into its mode of onset, duration, exact location, and its relation to the bowel movement. The sensation may be one of discomfort, such as of pressure, weight, bearing down, or a constant desire to defecate.

The pain in anal fissure is lancinating in character, is aggravated by the stool, and is marked by a time interval; that in anal ulcer, or chronic fissure, is usually described as a constant dull throbbing which is also aggravated by the passage of the stool. Protruding internal and thrombotic external hemorrhoids, as a rule, produce a prolonged dull aching pain. Papillitis, cryptitis, and perianal submucous fistula result in sphincter spasm and an aggravating aching pain. Uncomplicated hemorrhoids, polyps, and early malignant lesions, as mentioned above, do not produce pain except by pressure.

Protrusion: In questioning a patient about protrusion from the rectum, the individual quite commonly states that there has been a protrusion for several months which cannot be replaced. Subsequent examination usually reveals a thrombotic external hemorrhoid or simple skin tags. In some instances a physician, after but a cursory examination, will attempt to replace what appears to be a prolapsed, strangulated hemorrhoid when, in fact, he is dealing with an ulcerated thrombotic external hemorrhoid, and efforts to force it inside the anal orifice can only add to the patient's pain and accomplish nothing. If a lesion is covered by skin the pathology is "external"

in nature, and must not be considered a protrusion from the anal orifice.

Internal hemorrhoids, when they protrude, are soft and boggy. Polyps and hypertrophied anal papillæ are much firmer in consistency. Unless such lesions become strangulated and edematous, they can usually be replaced without difficulty. Prolapse and procidentia of the rectum are usually more or less uniform in the amount of protrusion. The patient should be questioned in regard to the character of the protrusion, the amount of bleeding, if any, and the relation to defecation, as well as the manner in which the protrusion can be replaced.

Discharge: Discharge from the anorectal region may consist of mucus, pus, feces, or debris, and may or may not be mixed with blood. The source of the discharge, from the anus or from an opening near the orifice, should be determined, as well as the duration, and whether it is continuous or intermittent. A mucoid discharge may be slight in amount, such as may be due to a small benign adenoma or papilloma, or may be profuse, leading one to suspect a villous tumor or mucous colitis. Purulent discharge from a site outside the anus is indicative of a fistula. Such a discharge from inside the anus suggests various forms of proctocolitis and malignancy with ulceration, in which cases the discharge will also contain blood, mucus, and debris. The discharge of a malignant tumor has a characteristic fetid odor.

Swelling: It is important to know the location and duration of any swelling in the anorectal area, and whether or not it is inflamed, tender, or movable. If rupture has occurred, the physician should know whether drainage was spontaneous or incisional. He should know if there has been a similar swelling at any time in the past. The most common cause of swelling in this area is, of course, an abscess. Cysts, lipomas, and other benign tumors are more rare.

Itching: Itching around the anus is quite common and is one of the most annoying symptoms with which one can be afflicted. It is usually worse at night and is aggravated by scratching. It is frequently intractable. Itching is, of course, merely a symptom. When it has been present for a long period of time and is accompanied by the characteristic changes in the perianal skin, however, we are justified in thinking of it as a disease in itself. In such cases careful investigation of all anal and rectal pathology must be made, for in the eradication of all such pathology lies the only hope of relief for these unhappy individuals. One of the most common etiologic factors in pruritis ani, in our experience, is a perianal submucous fistula, which is characterized by an anal ulcer, spasm of the sphinc-

ter, inflammation of the anal mucosa, and undermining of the anal skin and mucosa. Treatment of the itching will be of no avail unless all such pathology is first eliminated.

Bowel Habits: A study of the bowel habits of the individual is important. The regularity of the bowel movement, the consistency of the stool, its size, shape and color, and whether it is streaked or mixed with blood, mucus, or pus should be noted. Diarrhea demands consideration of the duration, character, and color, as well as of the number of stools per day. The diarrhea may alternate with constipation. Inquiry should be made into the use of cathartics and enemas. A "change in bowel habits," which means any variation from what may be considered normal or habitual for the individual in question, is a common symptom of cancer of the rectum and sigmoid.

Present History: The symptoms of many anorectal diseases are not confined strictly to that region of the body. For that reason, the physician will do well to consider any symptom or condition which may have either direct or indirect bearing on the pathology in question. In addition to such subjective findings as nausea, vomiting, anorexia, and loss of weight, the examiner should not overlook a feeling of weight or pressure in the pelvis, lumbosacral pain, or radiating pains in the legs. If one subscribes to the theory of "foci of infection," he would do well to remember that localized chronic infections, such as cryptitis, papillitis, and fistula are quite common and may produce symptoms far removed from the anorectal region.

Past History: Previous serious illnesses, surgical operations, or injuries, and, particularly, operations or injuries involving the anorectal area should be included in the history.

Family History: There are a number of diseases, the occurrence of which in one or more members of the family might have some bearing on the case at hand, which should be embodied in the history. These include: tuberculosis, cancer, polyposis, syphilis, and amebiasis.

The Proctologic Examination

The examination of the rectum may be divided into five distinct steps:

1. Inspection
2. Palpation
3. Digital examination
4. Anoscopy
5. Proctosigmoidoscopy

The first four of these steps require a minimum of time, effort and equipment. They can be performed by any physician and must be considered an integral part of every physical examination. The fifth step, proctosigmoidoscopy, requires more time, a certain amount of skill, and special instruments and equipment. It is inconceivable, however, that any surgeon who performs operations on the rectum and colon, or any internist or general practitioner who undertakes the treatment of ulcerative colitis, amebiasis, or other inflammatory conditions of the lower intestinal tract, should not be adept at this procedure and use it regularly in his work.

The position of the patient for rectal examination is of the utmost importance. Since, as was previously mentioned, many patients seek medical advice only because they are in pain, it is desirable that they be placed in the most comfortable position that is consistent with adequate exposure, good lighting, and freedom of movement for the examiner. In the opinion of most proctologists, that position is the Sim's, or left lateral. Should the examiner be left-handed, the right lateral position would seem preferable. In the Sim's position, the patient lies on his left side with the left arm behind his back and the left leg extended. The right leg is flexed at the hip and knee. The practice of having a patient bend over a table or chair for rectal examination is mentioned only to be condemned. This procedure may be acceptable to the urologist for prostatic massage, but is entirely inadequate for the diagnosis of anorectal pathology.

Inspection: With the patient in the left-lateral position and properly draped, the buttocks are gently separated, exposing the entire perianal region. At this time the examiner should note excoriations or redness of the skin, unusual prominence of radiating folds of skin, swelling, external hemorrhoids, condylomas, protrusions from the anus, or external openings of sinuses or fistulae. Inspection of the sacral area may reveal a pilonidal dimple or sinus.

Palpation: Each buttock is then gently palpated. The induration, tenderness, and fluctuation of an abscess is readily discernible. When inspection has revealed an external fistulous opening, the indurated tract can usually be felt leading in the direction of the anus.

Digital Examination: In this phase of the rectal examination the physician may quickly, though inadvertently, destroy the confidence he has thus far built up in his patient, and make further cooperation on the part of the patient almost impossible. Since, as was previously mentioned, a great many patients do not consult the doctor until they are in pain, it is essential that this portion of the examination be carried out as gently and as painlessly as possible. If the

physician is careless or rough in his digital examination, not only will that phase of his investigation be uninformative but instrumental examination will be impossible.

With the patient in the left-lateral position, and properly draped, the index finger of the right hand, gloved with a finger cot and well smeared with a water-soluble lubricant, is inserted into the anal canal. As the ball of the finger is placed against the anus, the patient is asked to gently bear down, as in the act of defecation, thus relaxing the sphincter and making introduction of the finger easy. With a slow rotary movement the finger is then inserted to its full length.

It is well to point out here that the vast majority of conditions affecting the rectum are within reach of the examining finger. A very important fact to remember is that 75 per cent of all malignancies involving the intestinal tract, exclusive of the stomach, are at or below the rectosigmoid junction and can be felt on careful digital examination. In patients presenting a suggestive cancer history it is well to have them rise to their left elbow, from the left-lateral to a semi-sitting position. Then, by asking them to strain down, an additional two inches of the bowel can often be palpated.

The examining finger should then be withdrawn with a circular motion, to detect any change from the normal smooth surface of the rectal mucosa. Uncomplicated internal hemorrhoids cannot be palpated since they are soft tumors lying upon a soft-tissue base. The experienced examiner may note a sense of fulness which will cause him to suspect the presence of such a condition. Tenderness or acute pain in the anal canal would suggest the presence of a fissure, ulcer, inflamed or ulcerated hemorrhoids, or, perhaps, a perianal submucous fistula. The prostate should be examined at this stage, as should the uterus, noting size, position, and mobility. The coccyx should also be examined with particular reference to its position and mobility, and whether or not motion causes pain. In the female, the rectovaginal septum can best be investigated with one finger in the rectum and another in the vagina. Valuable information is sometimes obtained in this way.

Particular attention should be paid to the tone of the sphincter muscle. Relaxation of this muscle is said to be one of the earliest symptoms of tertiary lues, but an atonic sphincter is also frequently seen in elderly and debilitated individuals. Sphincter spasm may indicate the presence of fissure, ulcer, cryptitis, papillitis, submucous abscess or fistula, or any other disease of an inflammatory nature.

Anoscopy: Any one of a variety of instruments may be used to visualize the anal canal and lower rectum. Most of the more com-

monly used scopes are electrically lighted but direct or reflected light may be used and is preferred by some men.

This portion of the examination should also be done in the left-lateral position. The instrument should be well lubricated and, with the right buttock retracted by an assistant or the patient, the tip of the scope is pressed gently but firmly against the anal orifice. As it enters the anal canal it should be rotated and inserted to its full length. The obturator is then removed and the light turned on. The mucosa of the lower rectum, anal canal and pectinate line is then inspected for hypertrophied papillae, inflamed crypts, internal hemorrhoids, internal openings of fistulous tracts, and ulcerations of the mucosa or skin.

Proctosigmoidoscopy: This procedure should be a routine part of the examination of every patient with rectal or colonic symptoms. All too often serious pathology is found just beyond the reach of the examining finger or the anoscope, even when disease in the lower rectum or anal canal apparently accounts for the symptoms presented by the patient. There is nothing to prevent a patient with a fissure or bleeding hemorrhoids from also having a carcinoma in the upper rectum or sigmoid.

It is essential that the bowel be thoroughly cleansed in order to perform adequate proctosigmoidoscopy. This is best accomplished by one or more cleansing enemas given from four to six hours prior to the examination. Some proctologists prefer to examine the patient before the bowel is cleansed, since valuable information is sometimes gleaned from the appearance, odor, etc., of the contents. In such cases, the examination must always be repeated after the bowel has been cleansed.

The position of the patient depends somewhat on the wishes of the examiner. A special proctoscopic table permits the patient to be placed in the inverted position and, in some cases, allows for easier passage of the instrument. In the absence of such a table, however, a very adequate sigmoidoscopic examination may be accomplished on any flat examining table by placing the patient in the exaggerated knee-chest position. Some very capable proctologists, in fact, prefer this position and, of course, it is available to all physicians.

The proctosigmoidoscope is a tubular instrument between six and twelve inches in length, and from one-half to one inch in diameter. It is electrically lighted, with the light at either the distal or proximal end of the tube. Most scopes are equipped with a lens at the proximal end which permits closing of the tube and allows for inflation of the bowel with air.

With the patient in the desired position and properly draped, the lubricated instrument is gently inserted through the anal canal. In so doing the instrument is "aimed" in a line running from the anus to the umbilicus. When the tip of the scope has entered the rectum, the obturator is removed, the light turned on, and the remainder of the procedure carried out under direct visualization. The instrument should never be passed blindly, since perforation of a diseased bowel wall may occur without any warning to the examiner.

In the absence of painful lesions of the anal canal, malignancies or strictures, it is usually possible to pass the instrument into the sigmoid without difficulty. In some instances, inflation of the bowel with air facilitates the passage of the instrument but this should be reserved for selected cases since it is not without danger and frequently causes considerable discomfort to the patient.

The examination of the bowel wall is usually carried out as the scope is withdrawn after inserting it to the highest level possible. The appearance of the mucosa is noted, especially inflammation and ulcerations. Polypi, malignancies, and diverticula may be readily detected. The valves of Houston should be carefully inspected, with special regard to the superior surfaces of these structures since an early malignancy in this location may easily be overlooked.

During this phase of the examination the opportunity for removing a section of tissue from all suspicious lesions should not be overlooked. Specimens should be removed through the sigmoidoscope under direct visualization by means of the specially designed alligator-jawed biopsy forcep. An effort should be made to include both normal mucosa and neoplastic tissue in each specimen sent to the pathologist.

Laboratory Procedures: A number of laboratory procedures are helpful, and at times indispensable, in making proctologic diagnoses. No effort is made to discuss them in the order of their value to the physician nor according to the frequency with which they may be used. Each has its place and, properly used, will add much information not otherwise obtainable. In no instance do these tests or examinations take the place of a careful history and a complete physical examination of the anus and rectum. They should be considered as supplementary aids—at times supplying the information needed to establish the diagnosis and, at other times, verifying the clinical impression obtained on examination.

Roentgenologic examination of the rectum is of little value and, indeed, is not needed since this region below the rectosigmoid is readily accessible to direct visualization and biopsy. X-ray study of

the colon by barium enema or by the double-contrast technic is a very important adjunct to our armamentarium. In my opinion, however, these examinations should be performed by a roentgenologist trained in this kind of work since an improperly performed or incorrectly interpreted x-ray study of the colon may give both the physician and the patient a false sense of security—with fatal results for the patient.

The biopsy has been previously mentioned. It should be used freely, not only in the uncertain or suspicious lesions, but also in the obvious malignancies, since grading of the carcinoma may often be of value in determining the proper procedure to follow in initiating treatment, and the prognosis.

The use of bacteriologic smears and cultures is known to all. In obtaining material for culture, the scope should be sterile when introduced and care should be exercised lest the sterile swabs be contaminated while being transferred from the tube to the scope and back to the tube. The use of these procedures usually requires the facilities of a well equipped clinical laboratory. Examination of stool for parasites and ova is invaluable.

The value of the following tests is obvious. The Kahn or Wassermann test for syphilis and the Frei test for lymphopathia venereum often give the clue that is necessary to establish the diagnosis in ulcerative lesions or strictures involving the rectum or anus. The benzedrine test is a common procedure for determining the presence of occult blood. Although tuberculosis of the anus and rectum is rare, it does occur and should not be overlooked. The tuberculin test is valuable but biopsy should also be used in suspected lesions since patients with pulmonary tuberculosis may have nontuberculous inflammatory lesions involving the anorectal region.

The place of the blood count and urinalysis in the study of a patient needs no emphasis.

It is apparent, then, that the examination of the anus, rectum, and sigmoid colon is a relatively simple procedure which can be performed by any physician, on any type of examining table, in a short period of time, and with a minimum of equipment. It should be a part of every physical examination.

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BIBLIOGRAPHY

1. Buie, L. A.: A Practical Proctology, Philadelphia: W. B. Saunders Co., 1938.
2. Bacon, H. E.: Anus, Rectum, Sigmoid Colon, ed. 3, Philadelphia: J. B. Lippincott Co., 1949.

APPENDICITIS IN THE LOUISVILLE GENERAL HOSPITAL*

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THIS is a report of the mortality and morbidity of acute appendicitis in the Louisville General Hospital. We will present a comparison of the mortality and morbidity rates of two periods. The first period covers the years 1937 through 1939, and was chosen to represent the prepenicillin era. The second period covers the years 1946 through 1948 when both sulfa drugs and penicillin were readily available for use.

The basis of the classification used in our series rested on the report of the pathologist and only cases reported as acute appendicitis in its different stages are in the series. Simple acute, acute suppurative, and gangrenous appendices are referred to as "noncomplicated cases." "Complicated cases" include acutely perforated appendicitis with or without abscess. There are 181 cases in the early series (1937-1939) and 167 in the recent series (1946-1948).

Methods of Treatment: Both before and after the advent of penicillin and sulfonamide therapy, an attempt was made in all patients to reestablish the electrolyte balance by the use of parenteral fluids when needed. In the later years there has been a more intelligent use of fluids and occasionally some blood has been used before operation in serious cases. We have never adopted a rigid routine of preoperative treatment. However, in later years it has been the tendency to give patients with suspected peritonitis a large dose of penicillin, usually around 100,000 units, and 2½ grams of sodium sulfadiazine before operation.

Primary appendectomy is done in all cases as soon as a diagnosis is made of acute appendicitis without localized abscess. In the presence of an acute peritonitis, operation is done as soon as the electrolyte balance is corrected and the patient has received his penicillin and sulfadiazine. We do not wait for localization in such cases.

All appendectomies are done through a McBurney or a similar muscle-splitting incision. In cases in which there is not a localized abscess no drains are introduced into the peritoneal cavity and the wound is closed tight to the fascia. Above the fascia, in some cases drains are inserted in the subcutaneous tissue, and in some the sub-

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cutaneous tissue and skin is left open and closed two or three days later.

Cases in which there is a localized abscess have always been treated conservatively. Since the days of penicillin the majority of these abscesses have resolved and an interval appendectomy has

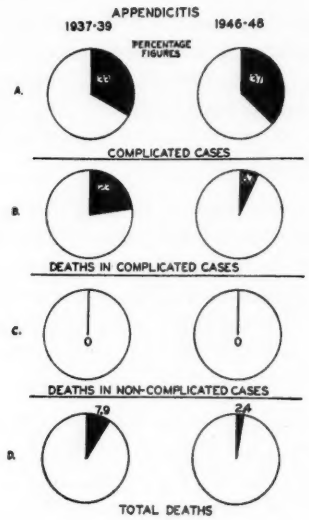


Chart I

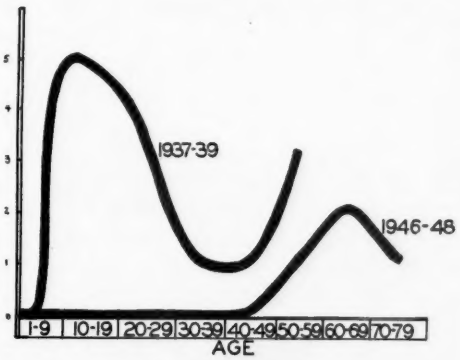
been done six weeks later. Before penicillin many of these abscesses did not resolve. In cases in which the abscess does not resolve the abdomen is opened through a McBurney incision and a drain introduced into the abscess cavity. The appendix is removed only if easily accessible. If the appendix is not readily accessible, a drain is inserted and an appendectomy is done approximately six weeks later.

In recent years penicillin has been given routinely to all cases of acute appendicitis after operation. An appendectomy is considered an unclean operation. In cases in which there is peritonitis or other extensive localized inflammation, penicillin or penicillin and sulfadiazine are used depending on the extent and degree of the inflammatory process.

Incidence of Perforation and Abscess Compared in the Two Series: In the earlier series 33 per cent of all cases were complicated. In the recent series 37 per cent were complicated (see Chart 1A). That this figure has remained essentially unchanged shows

that further efforts should be made to teach the public of the seriousness of abdominal pain and to report earlier for treatment. However, the closeness of the figures makes our mortality comparison more accurate.

Comparison of Mortality in the Two Series: The "noncomplicated cases" (see Chart 1C) (simple acute, acute suppurative, and acute gangrenous) accounted for no deaths in either series. In the complicated cases (perforation with or without abscess) death occurred in 23 per cent of the earlier series and 6.7 per cent of the recent series (see Chart 1B). It is in this group that the effects of the antibiotics are evident. The mortality in all cases in the first series was 7.9 per cent and in the recent series 2.4 per cent (Chart 1D).



DEATHS BY AGE

Chart II

Comparison of Mortality by Ages in the Two Series: When broken down according to age, a striking trend is noted. In the earlier series, death occurred at all ages with highest incidence in the younger groups, dropped in the middle years, and rose again in those 50 years old or more (Chart II). In the recent series all deaths occurred in patients 50 years or more. Any further efforts to reduce our mortality rate will have to be concentrated on the older age group.

Analysis of Recent Series: A detailed analysis of the recent series was made in an effort to evaluate our methods and results. The total series included 167 consecutive cases. Antibiotics and sulfonamides were available for use in these cases.

A. Age: The age distribution of our recent series (Chart III) was similar to that of other studies and to our previous earlier series (Chart IV).

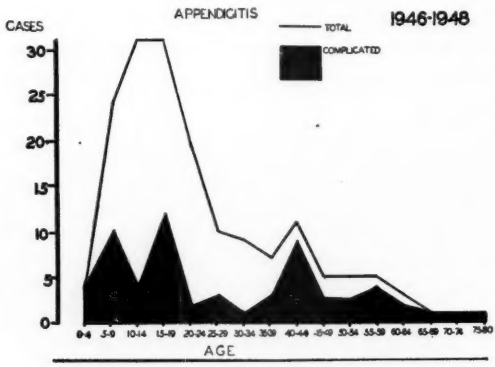


Chart III. Age Distribution 1946-1948

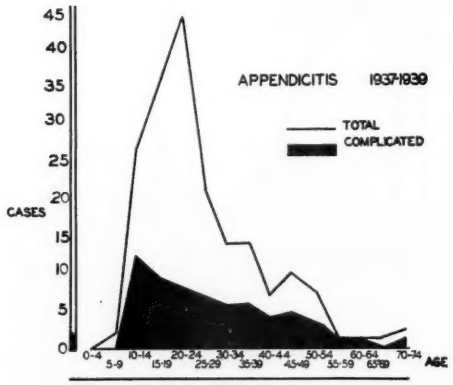


Chart IV. Age Distribution 1937-1939

In considering both groups the highest incidence was found in the group 10 to 20 years old. The youngest patient was 18 months old and the oldest was 75 years. In the earlier series the highest incidence of complicated cases was in the youngest age group (1 to 4 years) where it was 100 per cent (Chart IV) and was high in both series in the older age group (50 years or more) (Charts III, IV). The bizarre symptomatology present in these age groups leads to delayed or mistaken diagnosis and consequently to more complications.

B. Sex: There were 114 cases of appendicitis among the male admissions and 53 among the female admissions (Figure VB). When corrected against the total hospital admissions it is seen that

this difference is much more marked (Chart VI). The rate per thousand for the males was 10.0 and for the females 3.3.

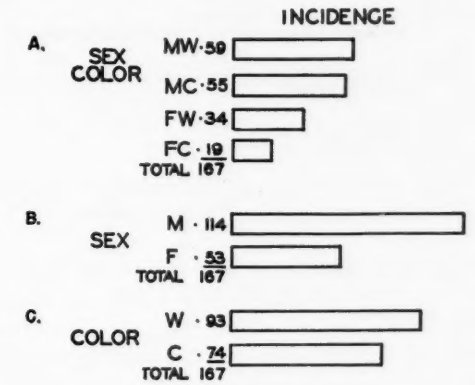


Chart V

C. Race: There were 93 white patients and 74 colored patients. When corrected against the total admissions to the hospital it gave a rate of 5.8 per thousand admissions for the white patients and 7.6 per thousand admissions for the colored patients (Charts VI, VII). The highest rate of incidence occurred in the colored males where it was 12.4 per thousand admissions. The lowest rate was in colored females where the rate was 2.5 (Chart VIII).

D. Appendicitis in the Colored Female: The apparent total incidence of appendicitis is much lower in the colored female than in

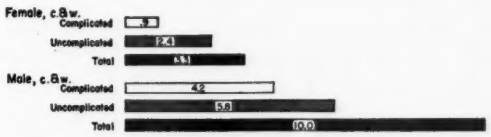


Chart VI. Sex: Corrected incidence per thousand admissions

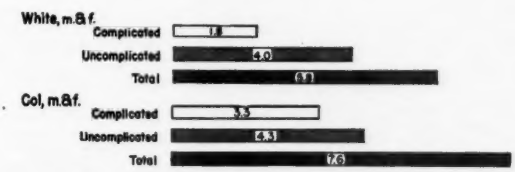


Chart VII. Race: Corrected incidence per thousand admissions

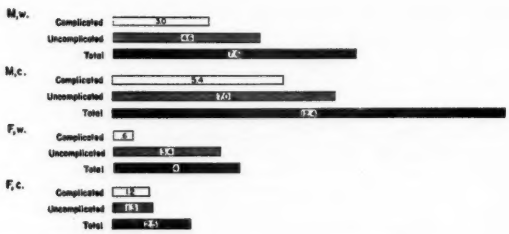


Chart VIII. Sex and Race: Corrected incidence per thousand admissions

the white female (Chart IX). We say "apparent" because we believe that the real total incidence in the colored female is as high as, if not higher than, in the white female. That there is not a racial immunity present is suggested by the high incidence in the colored male.

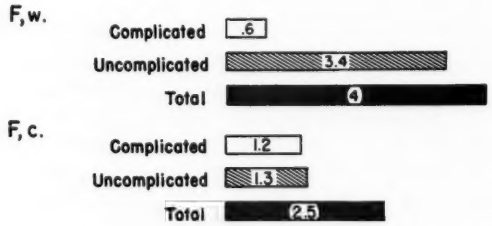


Chart IX. Race: Corrected incidence in females per thousand admissions

On further breakdown of these figures we found that the incidence rate of uncomplicated appendicitis in the colored female was much lower than in the white female, while the rate of complicated appendicitis was much higher (Chart IX).

The increase of complicated appendicitis in the colored female becomes much more striking when broken down into age groups (Chart X). In colored females below the age of fourteen 33 per cent of the appendicitis was complicated, which is about the same rate as for the entire group (37 per cent). In the group above fourteen, the age group where pelvic inflammatory disease is so common, 60 per cent of all cases were complicated.

We suggest that the high incidence of pelvic inflammatory disease in the colored female tends to mask acute appendicitis. It masks the disease from both doctor and the patient. Colored women who have been treated conservatively by the hospital clinics for pelvic

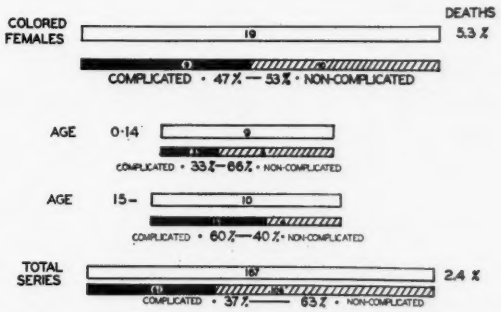


Chart X. Comparison of incidence: Colored females and total series

inflammatory disease may treat themselves at home for pelvic inflammatory disease with the previously recommended therapy when they actually have appendicitis. When they do come to the hospital with low abdominal pain undoubtedly appendicitis is often confused with pelvic inflammatory disease.

Thus, appendicitis in the colored female is likely to subside or go on to perforation and abscess formation under conservative therapy for the supposed pelvic inflammatory disease. We think that this accounts for the low incidence of simple, and the high incidence of complicated cases in our colored females.

The death rate of 5.3 per cent among negresses is over two times higher than that of the entire group. This is of course due to the high incidence of complicated appendicitis in this group of patients.

E. Duration of Symptoms (Chart XI).

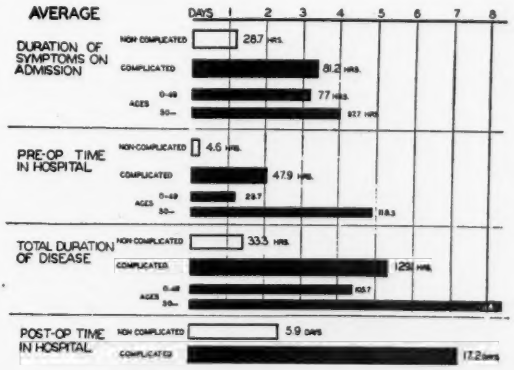


Chart XI

Preoperative Time: In the uncomplicated series the average duration of symptoms at the time of admission to the hospital was 28.7 hours. They spent an additional 4.6 hours in the hospital making a total duration at time of surgery of 33.3 hours.

In the complicated series the average duration of symptoms at time of admission was 81.2 hours. They spent an additional 47.9 hours in the hospital preoperatively, making a total of 129.1 hours (5+ days) at the time of surgery.

This is even more striking in the complicated cases of those 50 years or older. The average duration of the symptoms at the time of admission was 97.7 hours (3+ days). They spent an additional 119.5 hours (5 days) in the hospital preoperatively for a total of 217.2 hours (8+ days) at the time of surgery.

While part of the long preoperative time was due to bizarre symptomatology, mistaken diagnosis and occasionally unwise procrastination of the physician, this was a minor factor. The average of the preoperative time was greatly elevated by the time spent in those cases in which the physician deliberately and wisely delayed operation until the abscess became well localized or resolved.

Postoperative Time: The uncomplicated cases left the hospital in an average of 5.9 days. Complicated cases spent 17 postoperative days (Chart XI).

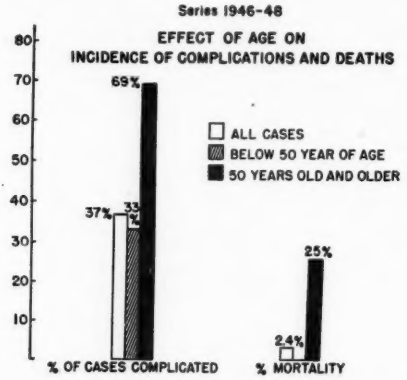


Chart XII

F. Deaths: There were four deaths in the series of 1946-1948 group for a death rate of 2.4 per cent. There were no deaths in the uncomplicated cases. All four deaths occurred in the complicated group (6.5 per cent). All of these patients were 50 years old or

older. Twenty-five per cent of all patients 50 years or older died (Chart XII). In only two of the four cases in which death occurred was the diagnosis made before death.

Two of the four cases (N.H. and A.L., Chart X) in which death occurred were correctly diagnosed. No diagnosis was made in one case (F. J., Chart XIII) and an incorrect diagnosis was made in the remaining case (W. P., Chart XIII).

DEATHS

	N.H.	A.L.	F.J.	W.P.
AGE	64	50	72	54
SEX · COLOR	F.C.	M.W.	M.C.	M.C.
DURATION OF SYMPTOMS — BEFORE ADMISSION	168 HRS.	12 HRS.	48+ HRS.	148 HRS.
DURATION OF SYMPTOMS — IN HOSPITAL PRE-OP.	288	3	DIED 23 HRS. AFTER ADM. NO SURGERY	42
DURATION OF SYMPTOMS — TOTAL	456	15	71+	190
PRE-OP. DIAGNOSIS	ACUTE APPENDICITIS WITH ABSCESS	ACUTE APPENDICITIS	DIABETIC COMA LEUKEMIA ABDOMINAL MASS	INTESTINAL OBSTRUCTION
SURG. PROCEDURE	DRAINAGE OF ABSCESS	APPENDECTOMY (PERFORATED)	NONE	APPENDECTOMY (PERFORATED)
COMPLICATIONS	PULMONARY EMBOLUS	HYPERTENSIVE C.V.D. CONGESTIVE FAILURE PERITONITIS	PERFORATED APPENDIX PERITONITIS SUBDIAPHRAGMATIC ABSCESS ATELECTASIS DIABETES BLOOD SUGAR 238	ATELECTASIS PULMONARY EDEMA APPENDICEAL ABSCESS PERITONITIS CARDIAC HYPERTROPHY PREVIOUS STROKE
POST-OP TIME	72 HRS.	144 HRS.	0	30 DAYS
AUTOPSY	YES	NO	YES	YES

Chart XIII

General Discussion

Chemotherapy in Appendicitis: The value of sulfonamide compounds and penicillin in traumatic surgery in preventing spreading fatal peritonitis was demonstrated during Warld War II. Practically every wounded soldier routinely received sulfadiazine and penicillin before and after operation. There is little doubt that this therapy made possible the relative infrequency of spreading fatal peritonitis.

We have not established a set routine for chemotherapy in the treatment of appendicitis either preoperatively or postoperatively. However, with very few exceptions all the patients with perforated or abscessed appendicitis received penicillin and sulfonamide in variable amounts. A small number received penicillin preoperatively.

There is a great amount of clinical evidence to indicate the effectiveness of chemotherapy in the control of peritonitis. A comparative study of sulfonamide drugs reported by Ravdin, Rhodes and Lockwood[1] reveals a distinct lowering of mortality and morbidity of appendicitis. The value of penicillin in traumatic injuries of the abdomen was demonstrated in World War II. Imes[2] reported distinct lowering of mortality and peritonitis with no instance of diffuse suppurative process during the Italian campaign. Drye[3] re-

ported a mortality due to peritonitis of less than 2 per cent in 3,200 abdominal wounds treated by the Second Auxiliary Surgical Group in the Mediterranean Theater of Operations and the European Theater of Operations. Crile[4] has stated that large doses of penicillin are necessary to control the peritonitis secondary to appendicitis and has reported no mortality in 50 cases so treated.

It is our contention that the use of antibiotics in the complicated cases has been a major factor in the reduction of our mortality rate from 7.9 per cent in 1937-1939 to 2.4 per cent in 1946-1948. Increased use of blood, intestinal decompression and more accurate administration of electrolytes also played an important rôle.

Further Reduction and Mortality: Further efforts to reduce mortality rates at the Louisville General Hospital call for an effort to lower the percentage of complicated cases by earlier diagnosis, more attention to the differential diagnosis of acute pelvic inflammatory disease and acute appendicitis, and concentration on the problem of the acute abdomen in the aged.

Lowering the percentage of complicated cases remains in the hands of the public health educators. The recent emphasis on the problems of cancer, tuberculosis, and poliomyelitis may possibly have caused neglect of appendicitis problems.

The conception of "It's P.I.D. until proved otherwise" when dealing with lower abdominal pain in the colored female should be abandoned.

Coexisting degenerative diseases contributed heavily to the fatal outcome. In the group 50 years or older none of the deaths could be attributed entirely and directly to appendicitis. However, we must still make a great effort to reduce the mortality by making earlier diagnosis, reducing the preoperative time by less procrastination and more active early surgical intervention, at times even without a definite diagnosis. We must also see the older patients much sooner.

Conclusions

1. The use of antibiotics and sulfonamides has markedly reduced the mortality in complicated appendicitis.

2. In patients over 50 years of age there is a long delay before admission and between the time of admission and operation.

3. There is an increase in the incidence of perforation and abscess with delay.

4. The mortality in the series in which penicillin and sulfonamides was used was limited to the patients over 50 years of age.

5. To reduce the incidence of perforation and abscess and the mortality rate particular attention must be paid to the patients over 50 years old. Operation must be done much earlier than heretofore. Definite clear-cut signs as seen in younger people should not be required before operation.

6. The incidence of appendicitis is probably about the same in the colored female as in the white female.

7. Appendicitis is frequently overlooked in the colored female and is often allowed to go on to spontaneous resolution or to perforation and abscess formation.

BIBLIOGRAPHY

1. Ravdin, I. S.; Rhodes, J. E., and Lockwood, J. S.: Results of Sulfonamide Prophylaxis in Surgery of Large Bowel, S. Clin. North America, *22:*1585-1591 (Dec.) 1942.
2. Imes, P. R.: Chemotherapy in Traumatic Surgery of the Abdomen, Ann. Surg. *123:*44-47 (Jan.) 1946.
3. Drye, J. C.: Penicillin and Sulfonamide Therapy in Abdominal Cases; Forward Surgery in the Severely Wounded, Second Auxiliary Surgical Group, pp. 197-202, 1945.
4. Crile, G. W.: Peritonitis—Appendiceal Origin—Treated with Massive Doses of Penicillin, Surg., Gynec. & Obst. *83:*150-162 (Aug.) 1946.

CARCINOMA OF ESOPHAGUS

A Report of Seventeen Cases*

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THIS report deals with 17 cases of carcinoma of the esophagus, the total number that have been treated at the Veterans Hospital, Louisville, Kentucky, since its opening in April, 1946. It is much too early to judge end results on the living cases; however, the series is representative and, we think, should be added to the growing literature. Several authors[1,2] have already very well summarized the evolution of surgical management of this disease since Adams and Phemister[3] in 1938 reported the first successful transthoracic resection and esophagogastrostomy for carcinoma to be performed in this country. The pertinent data in these cases are set forth in Tables I and II.

ETIOLOGY

No significant causative factors have appeared in any of the 17 cases here presented. Watson[4] listed certain congenital anomalies as predisposing to this condition. In a later article[5] he called attention to the relationship of the esophagus to the oral cavity and wondered if the same irritant might not produce similar changes in both locations. He listed fourteen instances in which primary cancer of the esophagus was associated with an independent primary intraoral cancer. One of our patients (No. 17, Table II) had an independent squamous cell carcinoma of the floor of the mouth. All sixteen of the squamous cell carcinoma patients had poor oral hygiene and were within the age group of squamous cell carcinoma of the oral cavity.

Six, or 35 per cent, of the seventeen patients were negroes, whereas only 13.5 per cent of our hospital admissions are of the colored race. Taquino,[6] in reviewing 145 cases from the Charity Hospital in New Orleans, found that 60 per cent occurred in negroes but pointed out that their colored admissions were close to that figure (45 per cent before the War; 55 per cent since). Leiter[7] in his excellent review of the literature found that the great preponderance of cases occurred in the white race.

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This paper is not intended to represent the views of the Veterans Administration but only those of the authors.

TABLE I

Clinical and Diagnostic Data on the Seventeen Patients with Esophageal Cancer

No.	Pt.	Age	Race	Symptoms & Duration	Wt. Loss	X-Ray	Esophagoscopy	Histology	Location and Extension
1.	P.P.G.	51	W.	Headaches & anorexia 8 wks., indigestion several years.	30 lb.	Pos.	Not done.	Squamous Gr. 2	Tracheal bifurcation. Extension: trachea, liver, lungs, skin, spleen, scalp, skull & ribs.
2.	B.S.S.	57	C.	Dysphagia & substernal pain 1 month.	Not recorded.	Pos.	Positive	Squamous Gr. 2	Tracheal bifurcation. Extension: trachea, aorta, heart, metastasis to regional nodes.
3.	J.M.	52	C.	Dysphagia 1 year, hoarseness, coughing & hemoptysis 2 weeks.	30 lb.	Neg.	Not done.	Squamous Gr. 3	Tracheal bifurcation. Extension: trachea, regional lymph nodes, supraclavicular nodes & lungs.
4.	L.B.	52	W.	Dysphagia 1 yr., pain in epigastrium 6 mos.	50 lb.	Pos.	Not done.	Squamous Gr. 3	Lower 3rd. Extension: right pleural cavity, regional lymph nodes & liver.
5.	E.H.B.	60	W.	Dysphagia & coughing uncertain duration.	Not recorded.	Pos.	Unsuccessful attempt.	Squamous Gr. 2	Upper 3rd. Extension: trachea, metastasis to right lung.
6.	W.S.	60	W.	Dysphagia, weakness, epigastric pain 7 mo.	60 lb.	Pos.	Lesion seen, 2 biopsies neg.	Squamous Gr. 3	Level of arch. Extension: pulmonary hilar & left gastric nodes.
7.	A.W.	53	W.	Dysphagia & weakness 10 months.	45 lb.	Pos.	Lesion seen. Biopsy pos.	Squamous Gr. 4	Level of arch. Extension: trachea, aorta, mediastinal and left gastric nodes.
8.	H.M.	67	C.	Dysphagia & weakness 2½ months.	47 lb.	Pos.	Lesion seen. Biopsy pos.	Squamous Gr. 2	Below arch. Extension: mediastinal nodes, cardiophrenic angle.
9.	R.C.	54	W.	Dysphagia & weakness 7 months.	50 lb.	Not done.	Not done.	Squamous Ungraded	Lower 3rd. Extension: to stomach, metastases to liver and kidney.
10.	C.McE.	55	W.	Dysphagia, weakness & substernal pain 2 mo.	25 lb.	Pos.	Lesion seen. Biopsy pos.	Squamous Gr. 2	Just below arch. Extension: no gross extension.
11.	W.Y.	52	C.	Dysphagia & epigastric pain 4 months.	29 lb.	Neg.	Lesion seen. Biopsy neg.	Squamous Gr. 4	Below arch. Extension: to bronchial, left gastric and pancreatic nodes.
12.	C.McD.	41	W.	Dysphagia & soreness in epigastrium 4 yrs.	20 lb.	Pos.	Lesion seen. Biopsy pos.	Adenoca. Gr. 3	Lower 3rd. Extension: to, but not beyond cardioesophageal line. No nodes involved.
13.	W.T.	62	C.	Pain in stomach & lt. flank & weakness 6 wk.	12 lb.	Neg.	Lesion seen. Biopsy pos.	Squamous Gr. 2	Just below arch. Extension: thoracic duct, mediastinal & left gastric nodes.
14.	E.F.	55	W.	Dysphagia & substernal pain 5 months.	20 lb.	Pos.	Lesion seen. Biopsy pos.	Squamous Gr. 4	Lower 3rd. Extension: mediastinal & lt. gastric nodes, also nodes left of aorta.
15.	G.P.	57	W.	Dysphagia, weakness, nausea & vomiting 2 wk.	54 lb.	Pos.	Lesion seen. Biopsy pos.	Squamous Gr. 4	Lower 3rd. Extension: to stomach & lt. pleura w/ extensive pleuritis.
16.	J.W.	59	W.	Dysphagia 3 years.	60 lb.	Pos.	Lesion seen. Biopsy pos.	Squamous Ungraded	Lower cervical. Extension: into trachea.
17.	M.A.	64	C.	Dysphagia & hoarseness 6 weeks.	22 lb.	Pos.	Lesion seen. Biopsy pos.	Squamous Gr. 2	Lower cervical. Extension: into trachea & involved rt. recurrent laryngeal nodes.

TABLE II

Treatment and Outcome (to Date) of Seventeen Patients with Esophageal Cancer

No.	Patient	Operation	Date of Op.	Prognosis at Oper.	Survival	Remarks
1.	P.P.G.	None. Inoperable.	----------	----------	Died 25th hosp. day.	Admitted terminally. Autopsy findings listed in Table I.
2.	B.S.S.	Inoperable. X-ray therapy at Hines Hospital May '46.	----------	----------	Died 4th hosp. day.	Admitted terminally. Autopsy findings listed in Table I.
3.	J.M.	None. Inoperable.	----------	----------	Died 7th hosp. day.	Admitted terminally. Autopsy done. See Table I.
4.	L.B.	None. Inoperable.	----------	----------	Died 3rd hosp. day.	Admitted terminally. Autopsy findings listed in Table I.
5.	E.H.B.	None. Inoperable.	----------	----------	Died 25th hosp. day.	Admitted terminally. Autopsy findings listed in Table I.
6.	W.S.	Resection. Esophagogastrostomy at level of arch.	7/14/47	Palliative	Died 7/15/47	Autopsy. Died of circulatory collapse. Slight hemopneumothorax.
7.	A.W.	Resection. Esophagogastrostomy at level of arch.	7/25/47	Palliative	Died 7/26/47 32 hrs. P.O.	Autopsy. Died of pulmonary congestion & edema & atelectasis left lower lobe.
8.	H.M.	Inoperable. Gastrostomy.	12/19/48	Palliative	Died 2/28/48 2 mo. P.O.	Had been transferred to another hospital. No autopsy reported.
9.	R.C.	Inoperable. Terminal. Admitted 2/24/48.	----------	Hopeless	Died 3/13/48	Autopsy. Extension to stomach & liver and metastases to kidney and peritoneum.
10.	C.McE.	Resection. Esophagogastrostomy above arch.	7/25/48	Curative	Living & well 11/24/49	No evidence of recurrence. No swallowing difficulties.
11.	W.Y.	Resection. Esophagogastrostomy above arch.	5/ 2/49	Palliative	Died 8/30/49 3 mo. P.O.	Autopsy. Metastases to liver & distant nodes. No swallowing difficulties.
12.	C.McD.	Resection. Esophagogastrostomy below arch.	11/29/48	Curative	Living & well 11/24/49	Considered inoperable, preoperatively. No swallowing trouble. Returned to work.
13.	W.T.	Resection. Esophagogastrostomy above arch.	2/ 2/49	Borderline	Living 11/15/49 w/disease	Wide extension at operation (see Table I). Now has positive sentinel node.
14.	E.F.	Resection. Esophagogastrostomy below arch.	4/ 6/49	Palliative	Died 8/25/49 4½ mo. P.O.	At operation: irremovable nodes. No swallowing trouble up to death. Died at home.
15.	G.P.	Inoperable. Terminal.	----------	----------	Died 54th hosp. day.	Autopsy showed wide extension to stomach, both pleurae, etc.
16.	J.W.	Inoperable. Gastrostomy.	8/ 8/49	Palliative	Died 8/17/49 9 days P.O.	Autopsy showed invasion of trachea w/terminal aspiration pneumonia. No other spread.
17.	M.A.	Inoperable. Invaded.	----------	----------	Died 33rd hosp. day.	No autopsy. Had concomitant squamous cell carcinoma of floor of mouth.

Symptoms

The symptomatology of the 17 cases is only significant in that one patient (No. 13, Table I) did not complain of dysphagia. Cancer of the esophagus may not reveal its identity to the patient or his doctor by specific complaints for a surprisingly long time or even until death. Thus, four of the present group (Nos. 2, 13, 15, and 17, Table I), all of whose lesions were advanced to incurability at admission, had symptoms of only two to six weeks' duration, surely a much shorter period than they must have been harboring their cancer. Also, one of us recalls a patient at the Boston City Hospital who died of unexplained hepatomegaly and cachexia, whose autopsy revealed a silent, small, pedunculated, squamous cell carcinoma of the lower esophagus and a metastasis-studded liver weighing 5,500 Gm.

If weight loss is significant of advanced disease, our group averaging 37 pounds under normal is certainly less hopeful than that of Payne and Claggett[8] which averaged 12 pounds weight loss. This is further borne out by the fact that of the 17 patients, in only two was operation curative (Nos. 10 and 12, Table I), and a third (No. 13, Table I) the prognosis was borderline since, although all grossly involved tissue was removed, the tumor had already invaded the thoracic duct and the periesophageal and left gastric nodes. It is interesting, furthermore, that the five patients surviving esophagectomy had lost only from 12 to 29 pounds, whereas those who were inoperable or died in the immediate postoperative period had lost from 30 to 60 pounds (see Tables I and II).

Physical Findings

Physical examination revealed nothing other than varying degrees of emaciation and dehydration in 15 of the 17 cases. Two patients (Nos. 1 and 4, Table I) were found to have liver enlargement from metastatic cancer. One of these had a positive sentinal node. Roentgen studies and esophagoscopy were carried out in all but one patient (No. 15, Table I), who had been explored at another hospital and who was transferred here for terminal care. It is noteworthy that in two instances barium swallow failed to reveal the lesion and that in one of these it was negative even after the cancer was localized and proved by esophagoscopy. This lesion, incidentally, measured only about 1.5 cm. in diameter and its owner (No. 13, Table I) had had definite symptoms for only six weeks, yet at operation, as described above, it was found to have already extended to the thoracic duct and below the diaphragm. These facts point to the importance of esophagoscopy in all patients with persistence of dysphagia or unexplained weight loss and symptoms that might

remotely point to the esophagus. This would be especially important in patients of cancer age.

Pathologic Behavior

Histologic study of this group revealed 16 squamous cell carcinomas and one adenocarcinoma. Of the 16 squamous cell carcinomas, seven were grade 2, three were grade 3, four were grade 4 and two were ungraded. It will be noted that one of the two curable cases was squamous, grade 2 (No. 10, Table I), and the other was adenocarcinoma, grade 3 (No. 12, Table I). This latter lesion involved the lower quarter of the esophagus, extending to but not beyond the cardioesophageal junction. Taquino[6] reports that, of 124 carcinomas of the esophagus, 17 were adenocarcinomas and that the great majority of these were in the lower third. The spread and metastasis of the presented lesions are as one would be led to expect from the literature.[9,10] We disagree with Garlock[2] who states that squamous cell carcinoma of the esophagus "rarely, if ever" metastasizes to the liver. In four of our patients (Nos. 1, 4, 9, and 11, Table II), as well as the patient we quote from the Boston City Hospital, the squamous esophageal lesion was found at autopsy to have metastasized to the liver. In general, growths involving the esophagus, below the aortic arch, tend to spread below the diaphragm and are relatively amenable to surgery. Those arising at the level of the arch or higher extend early to the pulmonary hilum and aorta and early become inoperable. The two patients with cervical esophageal growths (Nos. 16 and 17, Table I), and four others with involvement at the level of the tracheal bifurcation (Nos. 1, 2, 3, and 5, Table I), already had invasion of the trachea when first seen. This emphasizes the importance of bronchoscopy in esophageal cancer arising at or above the aortic arch.

Technical Considerations

The technic employed in our esophagectomies has not varied essentially from that described by Sweet,[11] Garlock[2] and others. We will, therefore, confine our remarks to certain points that appear important in the surgical management. Our experience in esophageal anastomosis includes, in addition to the seven esophagogastrostomies of this report, eight additional cases which are summarized in Table III.

Preoperative Preparation. The essentials of the preoperative preparation consist in:

a. Bringing the patient's red count, hemoglobin and plasma proteins to normal, or nearly normal, by adequate transfusion, high

TABLE III

Esophageal Anastomoses Other Than for Cancer of Esophagus

No.	Patient	Operation	Diagnosis	Outcome
1.	C.M.	Esophagectomy, Esophagogastrostomy below arch.	Multiple esophageal diverticulae. Advanced tuberculosis.	Died 5th P.O. day. Autopsy showed intact anastomosis. Bilat. pneumothorax and atelectasis.
2.	T.J.C.	Total gastrectomy, splenectomy, partial pancreactectomy. Esophagojejunostomy.	Adenocarcinoma of proximal stomach involving tail of pancreas.	Died of Ca. approx. 10 months postoperative. No swallowing difficulty.
3.	R.H.	Subtotal gastrectomy & partial pancreatectomy. Esophagogastrostomy.	Adenocarcinoma of gastric cardia involving tail of pancreas.	Died of Ca. 13 months postoperative. No swallowing difficulty.
4.	C.W.	Total gastrectomy & splenectomy. Esophagojejunostomy.	Adenocarcinoma of distal ¾ of stomach with involvement of nodes.	Living with disease 9/2/48. No swallowing difficulty noted.
5.	O.H.W.	Resection of distal esophagus and cardia. Esophagogastrostomy.	Traumatic diaphragmatic hernia with gangrene of cardia.	Died 30 days P.O. Autopsy showed intact anastomosis and pulmonary embolism.
6.	J.J.	Total gastrectomy, resection of spleen, distal pancreas & left lobe of liver. Esophagojejunostomy.	Advanced gastric cancer with repeated hemorrhages and a final massive one.	Died 48 hrs. postoperative. Anastomosis intact at this time.
7.	E.D.	Resection of distal esophagus, gastric cardia & left liver lobe.	Advanced portal hypertension with spleen already out.	Died 4 hrs. postoperative of uncontrollable hemorrhage.
8.	W.M.	Total gastrectomy. Esophagojejunostomy.	Multiple gastric polyposis with early malignancy.	Smooth convalescence. No swallowing difficulty.

protein intake and supplementary intravenous protein hydrolysates. It will be found that the patient's general nutrition will keep pace with this intensive therapy.

b. Management of the local lesion. Frequent irrigations are made through a Levin tube followed by local instillation of small amounts of sulfasuxidine. Likewise, for two or three days preoperatively the patient is placed on parenteral penicillin and oral streptomycin.

Operation. Adequate exposure is absolutely essential, especially when high anastomoses are to be performed. We have been satisfied with a long incision through the bed of the resected seventh rib with additional subperiosteal, paravertebral resections of short segments of the sixth, fifth, and fourth ribs, as needed. The double thoracotomies through the bed of the resected seventh or eighth and fourth ribs proposed by DeBakey and Ochsner[12] might be of advantage in extremely high anastomoses. In the esophageal resection itself, we attempt, as far as possible, to remove in a single block of tissue the esophagus and surrounding lymph nodes. We also believe that, if the periesophageal nodes are involved with cancer, the subdiaphragmatic and left gastric nodes and a rim of esophageal hiatus should be removed in continuity with the intrathoracic block dissection. It has seemed advisable routinely to expose and protect the thoracic duct. In one patient (No. 13, Table II), who had a nodule of carcinoma within the thoracic duct, this structure was resected with the contained nodule, and the proximal and distal ends were carefully ligated. No demonstrable ill effects followed.

It is of no particular moment if the right pleura is opened during the operation. However, a portable chest plate should be taken before the patient leaves the operating table and both pleural cavities should be aspirated free of air and fluid.

As to the anastomosis itself, we have used in all cases a double row of interrupted 000 cotton sutures, the inner through all layers, the outer row of inverting Cushing or Lambert type sutures. It has not appeared necessary to make the circular hole in the stomach proposed by Sweet[12] or the extra vertical incision in the esophageal stump as proposed by DeBakey and Ochsner.[13] We simply make a transverse incision in the stomach of the same width as the esophageal stoma. This is made, of course, after the back row of sutures between esophagus and stomach has been placed. In none of our fifteen anastomoses has there been any demonstrable leak, stricture or swallowing difficulty. However, four of the fifteen died within the first forty-eight hours postoperatively, thus scarcely allowing time for any anastomotic defect to appear.

One of the most important steps in the entire procedure is the adequate suspension of the stomach within the chest so that there is absolutely no tension at the suture line. The animal experiments of Carter, Stevenson and Abbott[14] have very nicely demonstrated this principle.

In closing the diaphragm, the surgeon should tack it loosely around the stomach with multiple fine sutures so as neither to constrict it in any way nor to strangulate the right gastric and gastroepiploic arteries which are the lifeline of the intrathoracic fundus.

Postoperative Care. There is surprising lack of unanimity among surgeons as to the advisability of an indwelling Levin tube and as to the postoperative limitation of oral intake. It is becoming increasingly apparent that if the anastomosis has been well performed the resumption of swallowing may be relatively early and that it matters little whether or not the Levin tube is used. It has been our custom, thus far, to leave a Levin tube down for the first four or five postoperative days and to interdict swallowing as much as possible during this period. However, Garlock[2] cautions against it and McNeer[15] believes it not only is not necessary but contributes to pulmonary complications.

Chemotherapy. We have had no case of empyema or wound infection, and attribute this as much to the maintenance of normal or nearly normal red count, hemoglobin and protein metabolism as to chemotherapy. We have of late, in extensive resections in contaminated fields, been dusting raw surfaces once or twice during the operation with an ampule or two of crystalline penicillin, reasoning that, even though much will be floated away in the bloody ooze, a high local concentration is created before bacteria have been allowed time to incubate. Postoperatively, for the first few days, large parenteral doses of penicillin and streptomycin are given.

Summary

1. Seventeen cases of carcinoma of the esophagus are presented, seven of which underwent resection with esophagogastrostomy.

2. No leakage or stricture has occurred in any of these or in eight other esophageal anastomoses performed by the authors.

3. The first two of the seven esophagectomies of this series died within thirty-six hours postoperative. The last five consecutive cases have survived operation. The two patients in whom resection with hope of cure was performed are living and well seventeen and twelve months, respectively, following operation. The patient with borderline curability is still alive, but with metastatic cancer, nine months following operation.

BIBLIOGRAPHY

1. Sweet, R. H.: Carcinoma of the Esophagus, J.A.M.A. *135:*485-490 (Oct. 25) 1947.
2. Garlock, John H.: Surgical Treatment of Carcinoma of the Esophagus and Upper Stomach, Surgery *23:*906-911 (June) 1948.
3. Adams, W. E., and Phemister, D. B.: Carcinoma of the Lower Thoracic Esophagus, J. Thoracic Surg. *7:*621-632, 1938.
4. Watson, W. L.: Cancer of the Esophagus: Clinical Observations of a Series of 930 Cases, Connecticut M. J. *6:*959-961, 1942.
5. Watson, W. L., and Pool, J. L.: Cancer of the Cervical Esophagus, Surgery *23:*893-905 (June) 1948.
6. Taquino, G. J., and Joseph, G. F.: Carcinoma of the Esophagus: An Analysis of 145 Cases with Special Reference to Metastases and Extensions, Ann. Otol., Rhinol. & Laryng. *56:*1041, 1947.
7. Leiter, E.: The Surgical Management of Carcinoma of the Esophagus, Surg. Staff Meet., V. A. Hosp., Des Moines, Feb. 2, 1949.
8. Payne, J. H., and Claggett, O. T.: Transthoracic Gastric Resections for Lesions of Cardia of Stomach and Lower Part of the Esophagus, Surgery *23:*913-920 (June) 1948.
9. Sweet, R. H.: Surgical Management of Carcinoma of Mid Thoracic Esophagus, New England J. Med. *233:*1-5 (July 5) 1945.
10. Churchill, E. D., and Sweet, R. H.: Transthoracic Resection of Tumors of Esophagus and Stomach, Ann. Surg. *115:*897-920 (June) 1942.
11. Sweet, R. H.: The Treatment of Carcinoma of the Esophagus and Cardiac End of the Stomach by Surgical Extirpation, Surgery *23:*952-975 (June) 1948.
12. Sweet, R. H.: Carcinoma of Midthoracic Esophagus, Ann. Surg. *124:*653-666 (Oct.) 1946.
13. DeBakey, M. E., and Ochsner, A.: Subtotal Esophagectomy and Esophagogastrostomy for High Intrathoracic Esophageal Lesions, Surgery *23:*935-951 (June) 1948.
14. Carter, B. N.; Stevenson, J., and Abbott, O. A.: Experimental Esophagogastrostomy, J. Thoracic Surg. *10:*446-474, 1941.
15. McNeer, G.: Principles Affecting Successful Esophageal Anastomosis, Surgery *26:*5, 1949.

THE SURGICAL RELIEF OF VESICAL NECK OBSTRUCTION IN CHILDREN*

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LESIONS of the vesical neck and posterior urethra in infants and children are characterized by their devastation to the upper urinary tract without symptoms or with only minimal symptoms. It is the rule rather than the exception that irreparable urinary tract damage precedes marked symptomatology. We have found it not uncommon that the child is first seen because the mother has noted an abdominal tumor which has been preceded by urinary frequency that has been disregarded or minimized. It will be noted from the cases presented that the instances of early diagnosis have been made by thorough urologic study.

To avoid the inexcusable tragedy of late diagnosis there are a few simple rules that will assist in circumventing this occurrence.

1. A complete urologic diagnostic study in every instance of continuous or intermittent pyuria whether gross or microscopic of only a few pus cells in a centrifuged catheterized urine.

2. An estimation of residual urine in any child or infant with urinary irregularity or frequency. A simple way to stimulate an infant to void is to introduce a catheter to the external vesical sphincter and quickly withdraw it. This will usually incite voiding and then the catheter is inserted into the bladder to obtain a true picture of residual urine. If the retained urine exceeds 10 c.c. the child should have a careful urologic study to determine the presence or absence of vesical obstruction. This study should include retrograde pyelograms with ureterograms.

3. A urologic study in any child complaining of or demonstrating gastrointestinal symptoms for which there is no satisfactory explanation.

4. Urologic investigation in every child or infant with an unexplained lower abdominal mass or tenderness.

Obstructing lesions of the vesical neck can be classified into those of the posterior urethra and those of the vesical neck proper. The urethral lesions are usually urethral valves which occur in the posterior urethra and characteristically permit the catheter to enter the bladder unobstructed and still voiding is obstructed. The vesical

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neck lesions effect a narrowing of the bladder outlet by bladder neck hypertrophy, hyperplasia or fibrosis. The common denominator in all these congenital lesions is obstruction.

These urethral and vesical neck lesions have in the past been approached either transvesically or transurethrally and neither method has proved entirely satisfactory in our hands. The transvesical approach characteristically lacks adequate visualization and positive hemostasis in vesical neck pathology. The transurethral approach has certain imposed mechanical limitations particularly in infants. We are aware of their apparent satisfactory use in some clinics, but our experience has not been entirely without dissatisfaction. We have adopted the retropubic approach in dealing with these lesions and have found this surgical route to possess both adequate visualization and positive hemostasis.

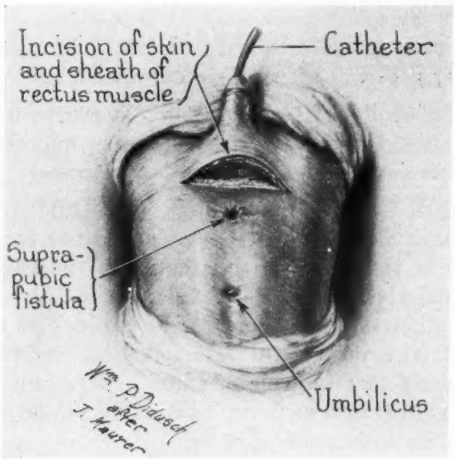

Fig. 1. Previous cystostomy drainage does not contraindicate the retropubic method for the relief of posterior urethral and vesical neck pathology.

The prostate and vesical neck is approached through either a longitudinal or transverse suprapubic incision which includes both the skin and rectus fascia. Infants of less than a year of age, without previous cystotomy drainage, we have used the longitudinal incision since it affords a more adequate exposure. Previous cystostomy does not prevent the use of this method in dealing with the vesical obstruction (fig. 1). Following the skin and fascial incision the rectus muscles are separated, the hypogastric fascia opened and the empty bladder displaced posteriorly and cephaled. The fat pad over the prostate and vesical neck is displaced laterally

to expose the variable vascular endopelvic fascia overlying the prostate. If the veins are prominent they are individually doubly ligated and severed. Endopelvic veins that are not prominent are disregarded and dealt with individually when bleeding ensues at the time of prostatic incision.

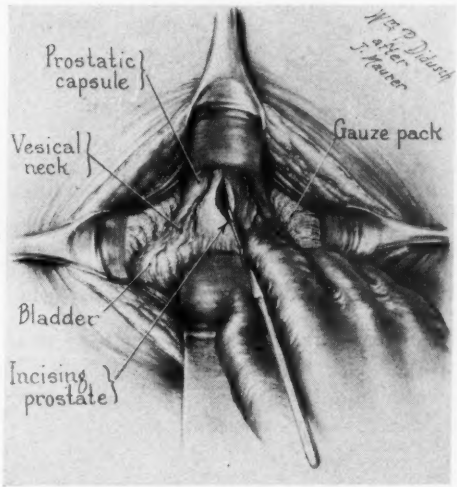

Fig. 2. The prostate is incised longitudinally from the apex to the prostatovesical junction.

The prostate is incised longitudinally extending from the apex of the gland to the prostatovesical junction and the incision is carried into the posterior urethra (fig. 2). At the superior angle of the incision the lateral branches of the prostatic venous plexus are divided and these vessels are ligated with a single suture on each side of the incision. These sutures are left long to act as tractors and when lateral tension is exerted the entire posterior urethra and vesical neck are adequately exposed for visualization and palpation (fig. 3). We mention palpation particularly since in two instances where the vesical neck obstruction appeared visually to be relieved, we palpated some dense underlying tissue. This was removed and the vesical neck could then be visualized and palpated to be free of an obstructing tissue.

Following the removal of the tissue about the vesical neck (fig. 4) the bleeding points which occur at approximately 5 and 7 o'clock are ligated with a suture of fine catgut and immediately the wound is dry. The prostatic capsule is approximated with a running suture of fine chromic catgut after a catheter of small caliber is introduced

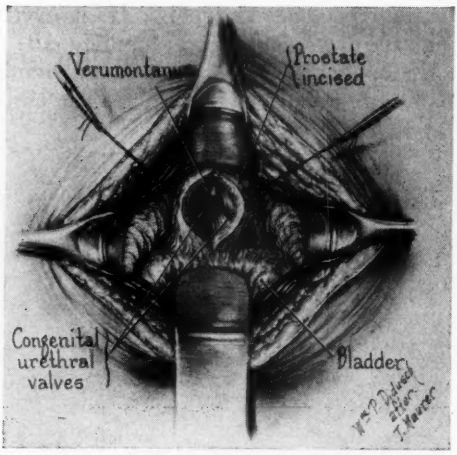

Fig. 3. Posterior urethral valves are visualized.

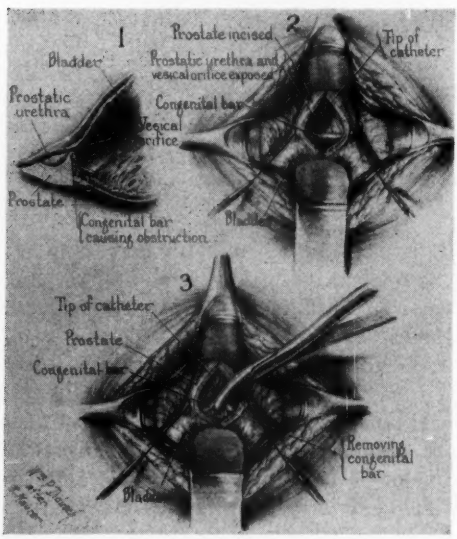

Fig. 4. Vesical neck pathology can be readily visualized and corrected.

into the bladder per urethram. The abdominal wound is closed in layers and a drain is left in place for two days. The catheter is removed on the fourth day and voiding in these children is spontaneous and uncomplicated.

We have treated five such children from three months to 13 years

of age with excellent results and a surprisingly insignificant morbidity. Upper tract damage was not evident in two children, 3 months and 9 months of age, since both these children were studied during periods of minimal symptoms and the obstructive uropathy found before extensive damage had taken place. The nine year old child had suffered irreparable renal damage in spite of the fact that he had been seen repeatedly by many physicians because of urinary disturbance of vague proportions.

All of the children in this group have had a most gratifying postoperative course. These children have been followed for more than two years in one instance, one year in another and the remaining three less than 9 months. In every instance there was no residual urine postoperatively and wound healing was primary in every instance except one in which there was a suprapubic fistula which closed after a urethral catheter was reinserted for four days.

Conclusion

The retropubic route to vesical neck and posterior urethral pathology in infancy and childhood is advocated. The method is discussed and a series of five cases is presented which demonstrated a most gratifying result in every instance. This numerically inadequate group of children is reported to suggest additional trial by others in an attempt at accurate evaluation of the method.

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THE NATURE OF AND TREATMENT OF TENDINITIS OF THE MUSCULOTENDINOUS CUFF OF THE SHOULDER AND SUBACROMIAL BURSITIS*

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Tendinitis of the musculo tendinous cuff of the shoulder and subacromial bursitis is a painful malady which is usually present in the supraspinatus or adjacent tendons, and may involve the bursa beneath the deltoid muscle in one or both shoulders. The pathology of the condition is poorly understood by most practitioners, and thus there are many forms of treatment, with the result that some patients sustain a distressing and severe disability as the result of inadequate treatment.

Confusion in therapy of tendinitis of the musculotendinous cuff of the shoulder and subacromial bursitis arises also from the fact that the bursitis has been attributed to a number of causes. The most accepted etiologic factor is thought to be the "wear and tear" of repeated trauma to the musculotendinous cuff of the shoulder, causing a degeneration of the tendinous fibers with at times a resulting calcium deposit in the supraspinatus tendon or adjacent tendons beneath the subacromial bursa. Later the calcified deposit may or may not rupture into the subacromial bursa. There are some types of bursitis in which no calcium deposits appear on x-ray, and in these the symptoms are attributed to degenerative changes resulting from injuries to the supraspinatus tendon.

In reviewing the statistics of several hundred patients treated it was found that as a rule the condition appears past 40 years of age, but it can occur between 20 and 40 years. Rarely does the condition occur under 20 years of age. Statistics from private practice show the condition occurs ten times as often in females as in males. It may be that women are more prone to seek medical care, and for this reason the condition comes to the attention of the physician in the female more often than in the male. The male is busily engaged in earning a living and cannot afford oftentimes to seek medical care for mild bursal symptoms.

Tendinitis and subacromial bursitis can be divided into three stages in order to describe the symptoms and physical findings more clearly. They are the acute, subacute and chronic phases.

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The acute tendinitis, or subacromial bursitis, presents the picture of a very painful shoulder which comes on over a period of 24 hours with early pain localized about 6 inches down from the shoulder

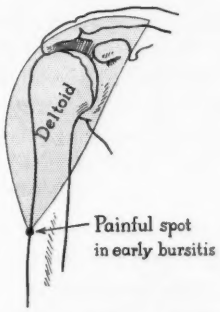

Fig. 1. Diagram of shoulder and arm showing painful spot in early bursitis.

joint in the region of the insertion of the deltoid muscle. However, on making pressure over the greater tuberosity of the humerus, tenderness there can be elicited at this early stage. From the region of the deltoid tubercle the pain usually spreads up the humerus and then involves the shoulder region. The patient may awaken with pain in the morning after having had a good night's sleep. Others describe the pain as having come on following a first gardening attempt of the year, shoveling snow, or some unusual exertion of the upper extremities. Others say the pain comes on following athletic events to which they are not accustomed, or following rest in bed where they have assumed some unusual position in sleeping. Some patients date the beginning of their pain to some minor physical effort. Shoulder pains are followed by spasms of the muscles which fix the shoulder joint at the side of the chest.

There are a few cases which develop pain in the shoulder slowly over a period of weeks, and sometimes months; they develop gradual stiffness in the shoulder. The involved shoulder may become swollen, and at times there is reddening of the skin over the painful area. The patient maintains the arm close to the side in adduction and internal rotation in an effort to splint the shoulder and prevent motion. Frequently the pain is so severe that the patient is unable to sleep, and only large doses of opiates can relieve the symptoms.

The acute case usually consults a practitioner about the condition and some form of heat is given with sedation. There are other patients whose shoulders are not quite so painful, and their symptoms are characterized by episodes of pain when the arm is placed

in certain positions; and like other conditions the shoulder is painful at night or on usage. This type of case, because of lack of acuteness, may not consult the physician for a number of weeks, with the result that the shoulder becomes stiff due to holding the arm to the side.

On x-ray examination of the acute shoulder one may find an amorphous opaque mass in the region of the musculotendinous cuff of the shoulder which varies from a pinhead size to masses extending to 1½ inches in length to ½ inch in diameter. At times there are separate areas of opaque material seen on x-ray films. In other cases of acute bursitis there is no evidence of any opaque material seen at all by x-ray.

The subacute subacromial bursitis comes on after a gradual subsidence of the inflammatory area of the shoulder has occurred. Atrophy of the deltoid muscle and other muscles around the shoulder is seen. Frequently pains are present in the trapezius muscle in the suprascapular region which radiate down the inner aspect of the arm and over the ulnar aspect of the forearm. Limitation of the shoulder is evident and the patient maintains the arm to the side in internal rotation.

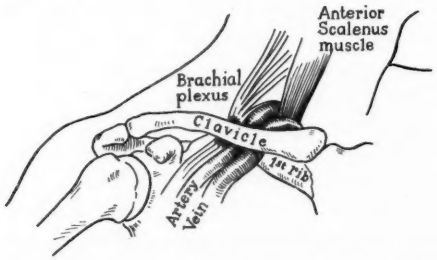

Fig. 2. Diagram illustrating the downward pull of an immobile shoulder in subacromial bursitis with branchial plexus compression causing pains radiating down into arm, forearm and hand.

As the condition passes from the subacute to the chronic stage the muscular atrophy of the shoulder is very marked, so that the head of the humerus is well outlined, and at times there appears to be some subluxation of the shoulder. Then the humerus is maintained against the side of the chest, and very little motion occurs in the shoulder joint. The motion of the shoulder region which remains is humeroscapular in type and this is even limited. Motion in internal rotation and external rotation is negligible. The patient develops greater pains in the suprascapular region radiating down the inner aspect of the arm, forearm and into the fourth and fifth fingers. This type of patient at times becomes addicted to barbiturates or opiates because of excessive usage for the chronic pain.

The pathologic changes seen in the subacromial bursa are usually due to the fact that there has been an injury to the musculotendinous cuff and frequently a calcium deposit has formed. Then with some unusual effort or enlargement of the calcium deposit it ruptures into the subacromial bursa. Frequently after this rupture occurs, acute symptoms become manifest in the shoulder, and a very red zone of reaction and inflammation is seen in the bursal sac. After the calcified deposit ruptures into the bursa, fluid is secreted and at times it has a yellowish look of thin pus. This is due to the fact that the bursal sac contains many leukocytes which are probably there to digest the calcareous material.

Fig. 3. Diagram showing the rupturing of calcium deposits into the subacromial bursa.

Following the rupture of the calcareous deposit a fibroserous-like material forms in the bursa, and the region becomes a dark red color with injection and thickening. After several weeks' time the acute process subsides, and in the meantime the inflammatory condition frequently invades the surrounding muscular structures and produces an acute and subacute inflammatory myositis due to the foreign body reaction. This inflammatory reaction at times is very intense with resulting bursal, capsular, and muscular adhesions and a consequent stiff shoulder.

The shoulder joint capsule becomes involved in the inflammatory process. If the patient holds the arm rigidly to the side for several weeks the shoulder joint capsule contracts and does not allow motion in the shoulder after being held in this position.

The tendinous deposits are usually found in the supraspinatus tendon, and there are a number which are found in the infraspinatus, subscapularis and teres minor tendons. The deposits from the supraspinatus tendon may be as large as an inch long and multi-

locular or just the size of an ordinary pea, to pinhead in diameter. The deposit consists of a whitish substance the consistency of an ointment. In the chronic cases this whitish material may be more gritty with fibrous strands running through it. Various chemical analyses of this material have been made, and they show a mixture of calcium oxalate and organic material, mostly fibrous tissue.

Fig. 4. Diagram illustrating use of an abduction splint for relief of pain and to prevent an adducted stiff painful shoulder.

As the subacromial bursitis becomes subacute and chronic, changes occur in the trabeculations of the bone underneath the bursa. There is atrophy of the bone in this region, and oftentimes a dark area in the x-ray film is present underneath the tuberosity. There is absorption of the lime salts from this region. At times there is bone atrophy of the entire humerus, acromion process of the scapula and clavicle. In some a spotty type of osteoporosis is seen. These pathologic bone changes are due usually to lack of usage of the shoulder and probably because of increased blood supply due to the inflammatory process.

The treatment of acute tendinitis or subacromial bursitis over a number of years has become very conservative. These patients lose a lot of sleep due to pain. The usual case requires sedation with opiates during the acute phase. There are some people who can get along with aspirin and small doses of codeine or barbiturates.

One measure that frequently relieves pain immediately is placing the arm on an abduction splint to get the extremity away from the side of the chest. Having a patient sit down on a chair beside a table and building up the arm with pillows to a position of 90 degrees

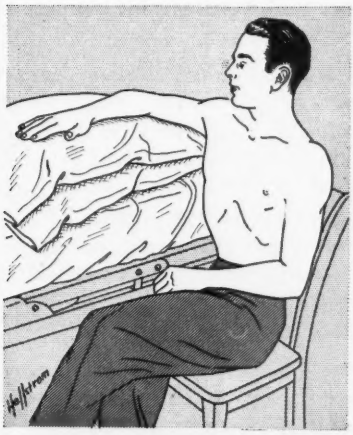

Fig. 5. Showing arm abducted on pillows of bed during treatment for subacromial bursitis.

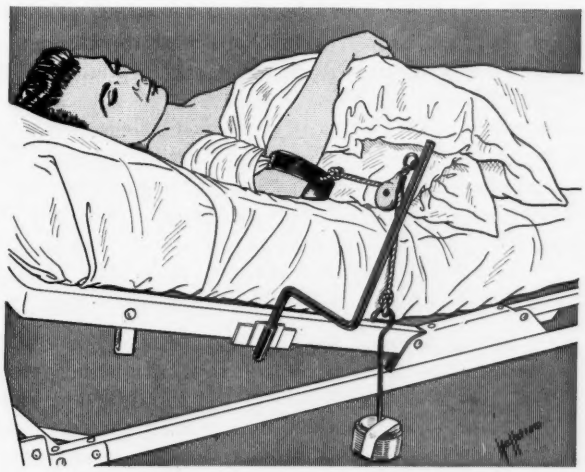

Fig. 6. Showing abduction traction in bed following irrigation or surgery for calcified deposits in tendinitis or subacromial bursitis.

away from the side of the chest is another method of abduction. This position frequently relieves pain altogether. A sling will help relieve pain, but in the end will produce harmful effects because it promotes the adducted position of the arm. When the patient has

a very acute shoulder with considerable redness then bed rest may be necessary for relief of pain.

The use of heat to the shoulder is very beneficial and should be used either if the patient goes to bed, wears an abduction splint or sits with his arm abducted on a table. In almost any case hot moist packs applied directly to the shoulder give great relief. One should place a hot water bottle over the hot packs, or keep them warm with an infra-red lamp. The hot pack is kept on 24 hours a day, and this is true especially in very painful cases. The shoulder is kept in abduction, either on a spint or on pillows except when certain exercises are taken.

In the mildly acute cases who want to continue at their occupation and be ambulatory, an injection of one or two per cent novocain can be made into the region of the bursa. After relieving the pain with novocain, we put the extremity through a complete range of motion, and then show the patient how to bend forward and take pendulum or circumduction exercises. These exercises should be done every hour by the clock when awake.

Another popular method of treatment of the acute subacromial bursitis with calcification is by aspiration and needling of the calcium deposit after injection of novocain. One finds the tender spot in the region below the acromion process of the scapula and greater tuberosity of the humerus. This area is injected with 20 to 30 c.c. of 1 per cent novocain. Then a needle of sufficient size or bore is introduced to puncture and withdraw any of the contents. At times 2 needles are inserted into the bursa for an irrigation process. Several introductions of the needle are necessary where the deposits are multilocular. This method is very useful when the deposits are soft and of a fluid type, and is less efficient in the chronic calcareous type. A shoulder manipulation should always follow the irrigation.

Treatment by irrigation is best carried on in a hospital because severe pain is usually experienced following the needling. For 24 hours an ice cap may be desirable over the shoulder and the arm is placed in abduction traction. Opiates are used for rest. After 24 hours then hot packs and the usual routine of exercising are begun.

In all cases one must see that they take pendulum or circumduction exercises every hour as soon as possible in order to increase the motion in the shoulder joint. These motions can be done very well after a few trials. Usually when the first 24 hours of severe pain has passed the patients can frequently do the exercises energetically. As soon as the shoulder has been moved with the pendulum motion the patients place the arm again in abduction, either on a splint or a pillow. While in this abducted position we try to have them put

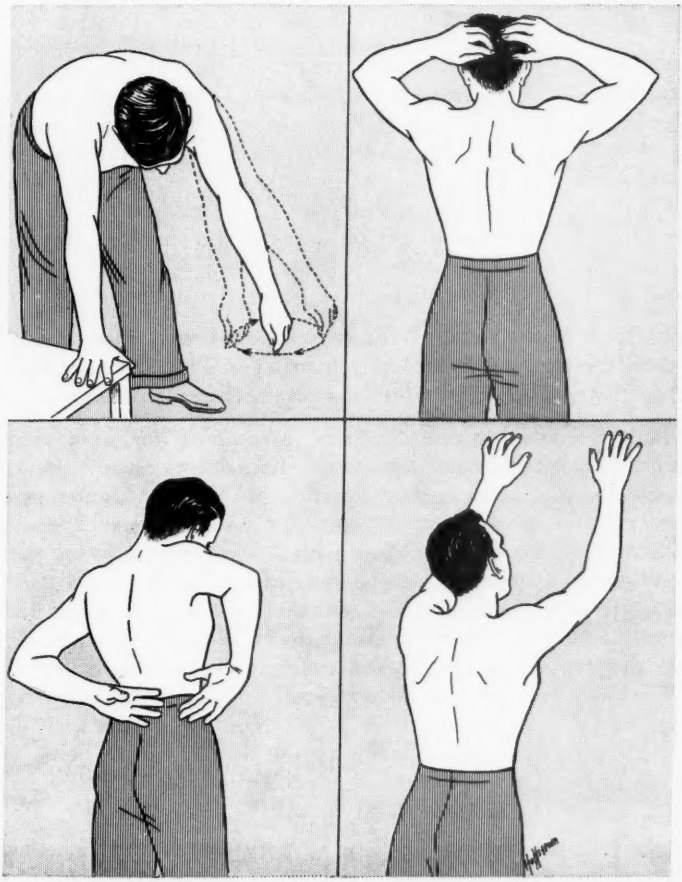

Fig. 7. Illustrations showing exercises to be used following all forms of treatment for subacromial bursitis; namely, pendulum and circumduction motions, external rotation stretching behind the head, internal stretching behind the back and wall crawling in elevation.

their hand behind their head in external rotation. On standing, following the exercises, they are urged to put their arm behind their back in internal rotation.

X-ray therapy in the mild to moderately acute cases has been found to be very effective. However, this is not the whole cure. Experience has shown again even with x-ray therapy it is necessary to apply hot packs, to abduct the shoulder and to use the exercises in order to obtain a good result. The use of x-ray therapy in the

subacute or more chronic cases has been found to be of very little value whatever.

Usually with from 5 to 7 days' treatment the patient's symptoms are quite well relieved and hot packs are no longer necessary. However, even though the pain is slight at the end of this time, the exercises must be continued together with sleeping with the arm away from the side on pillows or on an abduction splint. During the day while a man is working at a desk, we advise him to keep his arm away from his side and to get up and put the shoulder through a complete range of motion every hour. That is, to put the hand behind the head, behind his back and take circumduction exercises.

The treatment described for these acute cases must be carried out until the patient is absolutely pain free. The exercises must be used until a normal range of motion has been reestablished.

There are indications for surgery in some of the cases of acute tendinitis with subacromial bursitis. In patients who have large deposits of calcium as seen by x-ray immediate evacuation by surgery saves a lot of time and relieves pain. The acute symptoms can be cleared up completely in from 5 to 7 days' time, and the patient can return to a sedentary occupation. However, in a laboring man with acute subacromial bursitis it takes a number of weeks before he can safely do hard work. In patients having large calcium deposits surgery is more indicated than in the smaller deposits, because there is more foreign body material to be absorbed from the region of the bursa. At times as much as a tablespoon of the amorphous calcified material is found in the region of the bursa. Some large calcium deposits are never absorbed, and a painful area may remain in the region of the tendon and bursa.

As a rule the patient who has surgery for a calcium deposit is very gratified with the result. He loses the bursal pain immediately and has only the surgical pain due to the incision. At the time of surgery the shoulder is manipulated completely, and afterwards the arm is placed in an abducted position. Within 24 hours he is out of bed, taking circumduction exercises and putting his hand behind his head and behind his back.

Treatment in the subacute and chronic cases is a more difficult problem. Patients who have had subdeltoid bursitis for a period of 6 to 8 weeks usually have had their arms immobilized at their sides. They may or may not have a calcium deposit in the region of the subarcormal bursa. The usual patient who has his arm stuck down to his side and has had sedatives for 6 to 8 weeks without much relief is a very miserable person. Frequently the chronic bursal patients are addicted to opiates by the time the orthopedist sees

them. This type of patient can not tolerate very much pain, and it takes considerable wisdom and understanding to appreciate their pitiful plight. These patients are advised either to enter the hospital or to be near one where they can receive physiotherapy twice a day. They are informed that it will be necessary to undergo some pain in order to overcome this condition. We find that if they will take heat in the form of an infra-red treatment and hot packs in between the physiotherapy treatments the majority of them do quite well. They are started on pendulum exercises, taught how to keep their arms away from their sides by putting pillows on a table and then resting their arms on the pillows; or by starting off with an abduction splint with the airplane part collapsed as much as necessary to accommodate the arm. Then the physiotherapist gives them wall crawling and other exercises following the application of heat. By gentle stretching and massage over a period of weeks the shoulder motion can be increased and a great part of the pain can be lost.

In the subacute and chronic cases which have been going on for a period of weeks it sometimes takes 3 months of treatment to get the shoulder completely freed. As the shoulder loosens, the patients become more free of pain. Many patients are seen with pains in the side of the neck radiating down the arm and forearm into the little finger. This is all due to the fact that the arm is held to their side and the brachial plexus is being stretched downward by the immobile shoulder not being raised very much in the normal daily usage.

After the subacute and chronic bursitis patients have physiotherapy and exercises for a period of several weeks, and dense adhesions still remain around the shoulder, then at times one must manipulate the shoulder under an anesthetic. Here a complete range of motion is sought after by stretching the shoulder in all directions. However, one must be careful in doing shoulder manipulations because the humerus can be fractured especially where there are dense adhesions. After a manipulation the patient is placed in an abducted position with traction on his arm, and an ice bag placed on the shoulder for 24 hours. Following this the patient gets out of bed and starts exercising the shoulder. First, the pendulum exercise where the arm and shoulder hang forward. Then after that they are put back in bed, and an attempt is made to put the arm through a complete range of motion. As soon as possible these patients sit beside the bed and place their arm on an abduction splint or on a pillow on the bed. Then at hourly intervals they take their arm off the splint and take circumduction exercises. Heat in the form of an infra-red lamp is applied frequently or a continuous hot pack to the shoulder to help alleviate the inflammation and swelling

which occur following the manipulation. This type of treatment is carried out for 7 to 10 days in the hospital.

On dismissal from the hospital the patient is put in the hands of a competent physiotherapist for further stretching and manipulation of the shoulder, preferably twice a day. The attending physician should see these patients frequently to observe their progress and for guidance of the physiotherapist. One has to impress the chronic patient with the necessity of hourly exercises and heat routine at home. This type of patient takes from 6 to 8 weeks usually to get a satisfactory range of motion. After manipulation the shoulder has a tendency to stiffen up again unless one keeps trying to maintain the motion by exercises, heat and massage. At times in the chronic case it is necessary to wear an abduction splint for a period of 3 to 4 weeks; and one should always sleep with the arm away from the side. The patients are always cautioned that for a period of 6 to 12 months they must take exercises at regular intervals in the daytime to prevent further recurrence of this condition. Usually as the range of motion increases the pain leaves the shoulder, and the pain radiating down to the little fingers and up into the neck gradually disappears.

One finds that at times a patient will have a bursitis in one shoulder this year and get over it, and then will have a bursitis in the opposite shoulder next year. At times some people have minor symptoms of bursitis which we think are due to a small tear in the supraspinatus tendon. These patients have recurring attacks when they strain their shoulder, and are cautioned that they must do no type of severe labor or hard work with their shoulder such as gardening or golfing until all symptoms have passed.

There is another group of chronic tendinitis and bursitis patients who have calcium deposits in their shoulder that remain constantly painful and almost anything you do to them will not help them outside of surgery. Any case that has a calcium deposit present with pain from day to day may need surgery for relief. Some residual calcium deposits seen on x-ray are not painful and are symptomless. Among the older patients with calcium deposits one must be constantly on the lookout for superior lung lobe tumors that have persistent shoulder pain, not relieved by conservative or surgical bursitis treatment.

Summary and Conclusions

The nature of tendinitis and bursitis of the shoulder cuff is poorly understood by many practitioners which results in inadequate therapy and a number of disabled shoulders.

Repeated traumas play a large part in etiology and appear to account for the pathology. Statistically the condition affects females more than males.

Three stages of tendinitis and bursitis are described with variations.

A number of primary forms of treatment are described which should almost always be followed by sedation, abduction of the arm, warm moist packs, and the proper exercises. Abduction and exercises usually prevent the disabling frozen shoulder.

Addiction is frequent in chronic cases.

The physician must see the patient almost daily to rehearse the exercises and to impress the patient with the necessity of this.

Surgery is beneficial in some acute and chronic cases.

Superior lung lobe tumors in older patients produce at times shoulder pain simulating bursitis and are confusing when the calcium deposit is present in the shoulder region.

OBSTRUCTION OF THE GASTROINTESTINAL TRACT IN INFANTS

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EVEN though obstruction of the gastrointestinal tract in the newborn and in infants is a relatively uncommon condition, it does happen with enough frequency so that more and more emphasis should be and has been placed upon early diagnosis and surgical intervention. In the past it has been true that many obstructions have gone unrecognized and undiagnosed until the infant had reached the point where surgery was hopeless. The following conditions represent most of the commoner types of obstruction. An effort will be made to point out a few factors which may be of help in diagnosis and management of the problems:

CONGENITAL PYLORIC STENOSIS

Probably the most frequent reason for surgery within the first few days or weeks of life is congenital pyloric stenosis. Previously, the mortality from pyloric stenosis reached staggering heights; however, during the past several decades the mortality rate has dropped precipitously so that at the present, the rate should approach one per cent—or even less. There were several reasons to account for the previous high mortality: first, many cases of pyloric stenosis went unrecognized and undiagnosed for days, or even weeks, so that by the time the infant was hospitalized, he was frequently appallingly dehydrated and quite often ounces to pounds below the birth weight—making the infant a poor surgical risk at the beginning. Secondly, a factor which contributed to the mortality rate was that if and when accurate diagnosis was made the pediatrician and the surgeon in their enthusiasm to correct the problem would advise an operation much too soon. We now know that congenital pyloric stenosis is never an emergency. Days, or even a week, may be used to prepare these infants for surgery. An adequate period of time must be used to restore their electrolyte and fluid balance, as well as protein levels approaching normal.

Congenital pyloric stenosis is mentioned in a paper of this type only to emphasize the fact that it is never an emergency, and adequate time must be taken to prepare these infants properly.

DUODENAL OBSTRUCTION

Obstruction of the duodenum in the newborn and in the first few weeks of life presents an interesting problem. The etiologic factors

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involved in duodenal obstruction may be roughly grouped into two classes: the intrinsic and extrinsic factors. The intrinsic factor in duodenal obstruction has its beginning during the formation of the intestinal tract and begins somewhere between the fifth and tenth weeks of fetal life. During this time the intestinal tract from the pylorus downward passes through a so-called "solid stage," at which time the lumen of the intestine is filled with epithelial cells. These cells later coalesce and an open tube is again formed. If something happens during this formation of a patent bowel, then one of two types of obstruction may occur: one variety may consist of nothing more than a thin diaphragm which completely obstructs the bowel at that point; the other may be single or multiple areas of atresia.

The presence of complete obstruction in the duodenum from either a congenital diaphragm or atresia will manifest itself within a day or two after delivery. The infant will take several feedings and then begin to regurgitate. There are several symptoms which help the pediatrician and surgeon differentiate this condition from pyloric stenosis. In the first place, the vomiting occurs much earlier; i.e., in the second or third day of life, while, ordinarily, vomiting from pyloric stenosis may not come on until after a week to ten days. Second, the vomiting in this condition is not the projectile type of vomiting seen in pyloric stenosis; and third, if the obstruction is below the ampulla of Vater the vomitus will contain bile.

The diagnosis of obstruction at this point may well be made easily with the history and an upright roentgenogram of the infant's abdomen. The characteristic findings will consist of a moderately dilated stomach and, quite often, the dilated duodenum may be visualized. There will be little or no air in the remainder of the gastrointestinal tract. Barium should never be given by mouth to these infants who, if they are vomiting, will regurgitate and then aspirate it. The only barium that may be used in these newborns should be in the form of a small thin enema to help rule out incomplete rotation of the cecum. This latter condition will be discussed further later.

The actual surgical management of these infants with intrinsic lesions of the duodenum depends entirely on the condition found at the time of exploration; however, by far the more important considerations are early recognition of the pathology present and adequate preparation of the infant before surgery.

There are two main situations responsible for extrinsic obstruction of the duodenum. These occur most often in the second and third part of the duodenum and are due to incomplete rotation of the cecum and colon with partial to complete obstruction of the

second to third part of the duodenum. The other situation is the presence of congenital bands which produce angulation or actual compression of the duodenum so that obstruction necessarily follows. If the obstruction is complete, then the history and sequence of events will be identical with the intrinsic obstruction group; but if obstruction is incomplete, then the infant may be carried along for weeks, months, or even longer and be erroneously diagnosed as a "feeding problem."

The stage is set for obstruction of the duodenum secondary to incomplete rotation of the cecum during the period of the first six to ten weeks of fetal life. At this time the amount of the rapidly growing intestinal tract becomes too great for the future peritoneal cavity and extrudes into the base of the yolk sac. When growth of the future peritoneal cavity has provided room for the extruded intestinal tract, it is withdrawn and rotation of the colon begins moving in a counter-clockwise direction from the left iliac fossa. If something happens at this point so that rotation is not complete, then the cecum and ascending colon make an abortive effort to find attachment to the posterior peritoneal wall. Such attachment of the cecum and ascending colon, if it happens in the right upper quadrant, produces varying degrees of obstruction of the second or third part of the duodenum. This obstruction may be complete at the time of birth, may not become complete until weeks later, or may never become complete. Here again, depending upon the degree of obstruction, symptoms may make themselves apparent within days or weeks. If the obstruction is complete, the vomiting will come on within a day or so, will not be of the projectile type and the vomitus will contain bile. Examination at this time may show distention of the upper abdomen and rather marked tympany over the entire abdomen. A roentgenogram with the infant in the upright position may show findings identical with intrinsic obstruction of the duodenum. Administration of a very thin barium enema will show the position of the cecum and ascending colon. Ordinarily, incomplete rotation with the cecum and ascending colon in the left upper quadrant may not produce symptoms.

As mentioned above, some of these incompletely rotated cecums may go for months or years undiagnosed as such. We have seen in our group here a child almost eight years old who had been repeatedly diagnosed as a feeding and behavior problem and upon whom no previous barium enema had been done. The obstruction was almost complete but did allow enough food and liquids to seep through the narrow duodenal orifice to keep him barely alive. The skeletal, muscular and mental growth were markedly retarded so that at the time of establishment of the diagnosis the child appeared

more like a three year old in all of these respects. At the time of exploration, the duodenum was found to be tremendously dilated, measuring approximately 12 cm. in diameter. Following corrective surgery, the patient's growth and development was quite rapid.

The actual surgical technic involved in the correction of the defect is not within the scope of this paper but it is felt that a word of warning should be again given to the uninitiated. The operation of necessity is division of the congenital bands producing the obstruction. The entire cecum, ascending colon and what should have been the hepatic flexure are allowed to drop over into the left side of the abdominal cavity. There should never be an effort directed towards placing the cecum and ascending colon into their normal anatomic location in the right iliac gutter. When the congenital bands are divided and the entire colon rests in the left side of the abdomen and if all necessary congenital bands are divided, it will be found that the course of the duodenum from its second portion to the duodenojejunal junction is almost directly downward.

In an occasional case of incomplete rotation of the cecum, there will also be incomplete fixation of the mesentery to the terminal jejunum and ileum so that a segmental midgut volvulus, with the superior mesenteric vessels becoming the point of rotation, may occur. It is felt by some that if there is incomplete fixation of this midgut, it is permissible to attempt to fix this mesentery to the posterior parietes to prevent this segmental volvulus.

There will be an occasional case in which congenital bands form, or are found, in the area in which the duodenum and jejunum pass from their retroperitoneal position and which is marked in the normal abdomen by the ligament of Treitz. In this area imperfectly or incorrectly formed bands may cross the duodenojejunal junction and produce partial to complete obstruction. Here again, the train of symptoms will be identical with other forms of obstruction of the duodenum and the positive roentgen findings will depend entirely upon the degree of obstruction. At the time of exploration, the technic of release of the obstruction will depend upon the pathology found. In an occasional case, the only surgery that will need to be done will be simple division of bands that have angulated or compressed the bowel. On the other hand, situations may present themselves which will require more extensive surgery and an occasional case may require a side to side duodenojejunostomy in order to by-pass the obstructed area.

Obstruction of the Small Bowel

Apparently the most frequent site for congenital atresia of the small bowel is in the ileum, and again the changes which lead to

atresia happen within the fifth to tenth weeks of fetal life when the intestinal tract goes through its many changes to emerge as a hollow tube. Congenital atresia may assume all forms. There may be a single atresic area, or the other extreme may be present in which the entire jejunum and ileum will have the appearance of multiple small "link sausages" attached to a mesentery. The surgeon and the patient are both fortunate if the atresic area is a single one. If multiple, then resection of the involved portion of the small bowel may be out of the question because of the removal of such a large segment of small bowel.

Symptoms of atresia of the jejunum or ileum will be that of any other type of small bowel obstruction with the onset of vomiting and degree of distention depending entirely upon the level of obstruction. One should not be deluded into false security by the presence of a diarrheal type of stool in an infant who is apparently obstructed. These diarrheal stools that some infants have with complete small bowel obstruction are apparently nothing more than a type of starvation diarrhea.

Another congenital malformation which may lead to obstruction of the small bowel is the presence of a segment of reduplication of the small bowel. A duplication of the jejunum or ileum may be in the form of a blind pouch with no opening into the normal small bowel; it may be attached at the proximal or distal end, or at both ends. All of these duplications are anomalies that occur on the mesenteric side of the small bowel. If the duplication is a blind pouch, then secretion of intestinal juices may distend the blind pouch to such a degree that the normal small bowel coursing over its surface may be partially or completely obstructed. Here the symptoms produced will depend entirely upon the degree of obstruction and the level of obstruction. The diagnosis will not be obvious until the time of exploration. It has been felt by some that mesenteric cysts may have been the end result of an area of duplication of a bowel.

Obstruction of the Colon

Fortunately, congenital stenosis or atresia or the presence of congenital diaphragms are relatively uncommon conditions in the colon; however, there may occur an occasional case where the infant will develop symptoms of obstruction late. The infant will have had no meconial stools which in the presence of a patent anus should put the pediatrician and the nursing staff on guard within twelve to twenty-four hours after delivery. The more common obstructed area in the colon is an imperforate anus or the presence of a very

tiny sinus leading from a rectal pouch which is well up within the hollow of the sacrum. Imperforate anus is mentioned only to emphasize a point which may be of importance. I do not believe that an imperforate anus is an emergency. The absence of a normal anal opening should be recognized at the time of delivery or very shortly thereafter and when discovered at this early time, the newborn should not be given feedings. The introduction of water or formula or breast milk will greatly increase the bacterial flora of the intestinal tract and produce gaseous distention at too rapid a rate. If complete obstruction is allowed to continue, it is true that a rectal pouch which would have been inaccessible from the perineal approach may gradually be pushed downward within twenty-four to thirty-six hours so that it may be reached from below. The formation of a left lower quadrant colostomy necessarily increases the mortality and morbidity in these newborns and makes any type of secondary repair a much more formidable procedure from a mechanical standpoint. If vomiting begins before the rectal pouch has been moved down to a satisfactory level, it is wise to keep the stomach fairly empty with continuous suction through a small catheter. If no milk or formula has been given to the infant, then the aspirating catheter will remain open rather than being clogged repeatedly with curds from feedings.

Summary

An attempt has been made in the foregoing to mention some of the gastrointestinal anomalies that are occasionally met, even though many of the congenital defects are relatively uncommon in their occurrence. Any or all of them should be kept in mind in the newborn when it does not follow the usual course of events in regard to intake of formula, or other unusual developments within the first few days of life.

There has been no effort made to set out the actual surgical management of the situation that presents itself. Rather an effort has been made to stress prodromal symptoms, physical and roentgenologic findings. Careful preoperative preparation of these infants must be stressed time and again. Actually, the preoperative preparation is of much greater importance than the mechanical management of the defect present from a surgical standpoint. A newborn with an obstructive lesion and in whom the blood chemistry and protein values are unbalanced is a poor surgical risk. After careful restoration of these values, the infants tolerate major surgery remarkably well.

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THE SURGICAL TREATMENT OF PEPTIC ULCER COMPARING VAGOTOMY AND GASTRIC RESECTION

A Preliminary Report*

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IN the surgical treatment of peptic ulcer at the Veterans Administration Hospital, Louisville, Kentucky, both subtotal gastric resection and vagotomy, as reintroduced by Dragstedt,[1] are employed. As a matter of policy, all cases suspected of peptic ulceration are admitted to the Medical Service where diagnosis is established and an adequate medical regime is given a thorough trial. Cases are considered for surgery on the basis of intractable pain under medical management, repeated perforation, repeated hemorrhage, or organic obstruction. Surgery is also indicated in patients with peptic ulcers who, for good economic reasons, cannot properly follow medical treatment. In addition, gastric ulcers in which malignancy cannot be excluded are considered candidates for operation.

Since this hospital opened in April, 1946, 82 patients have been operated upon for peptic ulcer. There has been but one hospital death in this series, giving a mortality of 1.2 per cent. This was a 59 year old male, moribund from acute massive hemorrhage, who was explored as a last resort. There have been no deaths among the elective cases.

SELECTION OF SURGICAL PROCEDURE

The selection of the type of operative procedure for the individual peptic ulcer has not been easy nor has it followed a hard and fast or uniform pattern throughout. At first, those patients with high acidity and large quantities of night secretion were treated by vagotomy, although some presented moderate to marked obstruction. Then for a period following an unusually obstinate case of gastric retention in spite of combined vagotomy and gastroenterostomy, patients with duodenal ulcer and moderate to severe obstruction underwent subtotal gastrectomy. Late in 1948, encour-

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The subject matter expressed in this article is not intended to represent the views of the Veterans Administration but only those of the authors.

aged by the good results of Dragstedt,[2] Crile,[3] and others with combined vagotomy and drainage procedures, we have resumed this type of operation. In doubtful cases, especially those with lesser degrees of acidity and night secretion, we have followed Griswold[4] and Schoen[5] in using the response to histamine as one of the deciding points. Thus, we lean toward gastric resection in patients showing a relatively slight acid response to the Hollander[6] insulin test and showing a marked increase following histamine. All marginal ulcers have been treated by vagotomy. It is generally accepted that all gastric ulcers, if treated surgically, should be resected. Exceptions are those cases in which under medical therapy the ulcer disappears on x-ray within three weeks. We locally excised one small and innocent looking gastric ulcer, lying 2½ inches above the pylorus on the lesser curvature, which was found benign on frozen section, and carried out vagotomy. In this case the permanent section proved to be early scirrhous carcinoma. This case was then resected and the patient is now living and well at thirty-seven months without gastrointestinal symptoms or evidence of recurrence.

Vagotomy

The first vagotomies were of the transthoracic type, but more recently the abdominal route has been chosen. This permits local exploration as well as a complementary emptying procedure to avoid postoperative retention difficulties due to atonia of the stomach which occurs with vagotomy alone. Since July, 1946, 49 vagotomies have been performed and followed from one to thirty-six months, or an average of fifteen months. Of these cases, the 43 that have been followed three or more months appear in the comparative study; the remaining 6 are included only for survival statistics. Of this number, 12 were transthoracic (one later required pyloroplasty), and of the 37 remaining cases, 12 had abdominal vagotomy alone (one later required pyloroplasty), 14 had vagotomy with pyloroplasty, and 11 had vagotomy with gastroenterostomy. The last 22 cases have had simultaneous complementary drainage procedure. If technically feasible, pyloroplasty is preferred over gastroenterostomy because we feel that the drainage is more nearly physiologic.

Subtotal Gastrectomy

During the same period of time as that covered by the vagotomy series, 33 patients with peptic ulcer have been treated by subtotal gastric resection. Of this number, three were operated upon for acute hemorrhage with one death; 30 received elective surgery with no deaths. Twenty-four of the elective cases have been followed

sufficiently long to warrant analysis (3 to 37 months, or an average of 16½ months), and 6 were performed less than two months ago. Only the 24 elective cases which were followed over three months are analyzed. Although this is a small group, in which 50 per cent were for gastric ulcer and the remainder for obstructive duodenal ulcer, it was felt worth while to compare the subtotal gastrectomies with the vagotomies.

Comparative Study of Vagotomy and Gastrectomy

Age, Sex and Race: In the vagotomy series, the age varied from 21 to 61, the average age being 39.7. In the subtotal gastric resection series, the age varied from 29 to 58, with an average of 49. All were white males except for two colored patients in each group.

Duration of Ulcer: Among the vagotomy cases there were five with a history of ulceration of two years or less. The remainder had had their ulcers from two to thirty years. In the gastrectomy series there were five with histories of two years or less, and the remainder ranged from two to twenty-six years.

X-Ray Proof of Peptic Ulcer: Preoperative x-rays showed an ulcer crater in 34 and deformity of the duodenal cap in 9 of the vagotomy cases. In the gastrectomy group, 20 showed crater and 4 showed deformity. In all patients of both groups undergoing laparotomy the presence of ulcer was verified.

TABLE I

Location of Ulcers

Vagotomy—	(43 cases)	Gastrectomy—	(24 cases)
	Per cent		*Per cent*
Gastric	0.0		50.0
Duodenal	85.8		33.3
Gastric and Duodenal	4.6		16.7
Marginal	9.3		0.0

Location of Ulcer: The location of ulcer in both groups is shown in Table I. It will be noted that 50 per cent of the resected cases were for gastric ulcer.

Preoperative Findings Compared: In Table II are listed the significant preoperative findings in each group. It should be explained that by significant obstruction is meant obstruction as evidenced by x-ray. Significant bleeding means bleeding which is definite enough to produce gross hematemesis and/or melena. Severe disability implies heavy loss of time from gainful employment. It

TABLE II

Preoperative Morbid Findings

Vagotomy—	(43 cases)	Gastrectomy—	(24 cases)
	Per cent		*Per cent*
Significant obstruction	20.9		70.8
Intractable ulcer pain	100.00		100.0
Significant bleeding	58.0		82.2
Weight loss (20 lbs. or over)	30.2		45.8
Perforations	30.2		20.8
Disability:			
Mild	25.5		20.8
Moderate	39.4		29.1
Severe	34.8		50.0

is noted from Table II that in both series most of the patients were markedly incapacitated prior to surgery.

Postoperative Findings Compared: Table III shows the degree of postoperative improvement of the items listed in Table II. One of the two patients of the gastrectomy group who bled postoperatively had been drinking turpentine and sugar for relief of ulcer pain. There is a strong evidence that the two postvagotomy patients with recurrent ulcer had had an incomplete neurectomy. In one of these patients, reexploration thirty months after transthoracic vagotomy and three months after perforation of his duodenal ulcer revealed several cordlike strands around the subdiaphragm-

TABLE III

Postoperative Morbid Findings

Vagotomy—	(43 cases)	Gastrectomy—	(24 cases)
	Per cent		*Per cent*
Significant retention (by x-ray)	71.9		0.0
Significant vomiting	6.9		16.7
Significant bleeding	2.3		8.3
Weight gain (10 lbs. or better)	81.2		33.3
Perforations	2.3		0.0
Recurrent ulcer	4.6		0.0
Ulcer pain	4.6		4.2
Abdominal pain other than ulcer	4.6		20.8
Disability:			
None	81.2		58.3
Mild	13.9		25.0
Moderate	4.		4.2
Severe	2.		4.2

atic esophagus. One of these was seen on microscopic section to contain nerve fibers. Whether these represented the fine vagal branches, missed at the transthoracic vagotomy, or regeneration since vagotomy, is not clear. The other patient, whose vagotomy is considered a failure due to persistence of pain, vomiting and on one occasion a small hematemesis, has a strongly positive insulin test.

TABLE IV

Side Effects of Operation

Vagotomy—	(43 cases)	Gastrectomy—	(24 cases)
	Per cent		*Per cent*
Belching	9.3	Dumping Syndrome	16.7
Flatus	9.3	Weakness	41.6
Fulness	11.6	Fulness	37.5
Diarrhea	2.3	Diarrhea	0.0
Cardiospasm	9.3	Marked weight loss (20 lbs.)	4.2

Untoward Side Effects: The untoward side effects of the vagotomies and gastrectomies in this study are set forth in Table IV. It is noteworthy in the vagotomy series that fulness, foul belching, flatus and diarrhea, although encountered, were minor complaints and not incapacitating. Cardiospasm, however, has been an unusually frequent and severe side effect among our postvagotomy patients. Two required a left phrenic nerve crush for relief. In the other two the condition was mild and temporary. We have been unable to ascribe any definite cause for this complication. Three of the four followed thoracic vagotomy in which more of the vagus nerve was resected. Possibly an unopposed sympathetic tonus was partly responsible.

Psychiatric Evaluation: Because of the psychosomatic nature of peptic ulcer, 38 of the patients in the vagotomy series were evaluated preoperatively by the Psychiatric Service with the idea of predicting the probable benefit of the procedure and also of noting any change in personality in the postoperative period. Five of 38 patients in the vagotomy series who received psychiatric evaluation had no apparent psychiatric disease. The remaining 31 were found to have mild to severe abnormalities, including schizoid personality, passive dependency, hostility reaction, neurotic somatization, etc. There were seven in whom the psychiatrist felt that surgery would be of little benefit to the patient. Of these seven, four had a good result and three had fair results. So far, the psychiatric follow-up of these patients is incomplete.

Complications: The complications of each series are listed in

TABLE V
Postoperative Complications

Vagotomy (43 cases) ...	No.	Gastrectomy (24 cases)	No.
Atelectasis	12	Atelectasis	2
Pneumonia	3	Incisional hernia	1
Wound infection	1	Thrombophlebitis	1
Obstruction	2	Phlebothrombosis with pulmonary infarct	1
Pneumothorax	1		
Conjunctivitis	1		
Cardiac decompensation	1		

Table V. Surprisingly, the incidence of atelectasis is higher following vagotomy than following gastrectomy. This complication occurred five times after the 12 thoracic vagotomies and eight times after the 31 abdominal vagotomies. Following the abdominal procedure, the incidence was 25 per cent after the twelve with vagotomy alone and 21 per cent after vagotomy plus an emptying procedure, although this extra procedure added considerably to the operating time.

TABLE VI
Clinical Evaluation

Vagotomy—	(43 cases)	Gastrectomy—	(24 cases)
	Per cent		*Per cent*
Good	76.6		62.5
Fair or satisfactory	18.6		33.3
Poor or failure	4.6		4.2

Clinical Evaluation: The clinical evaluation of the two groups is compared in Table VI. Those cases classified as "good" are patients who are free of gastrointestinal symptoms and are gainfully employed. By "satisfactory" (or fair), we mean those patients who have no ulcer distress and the gastrointestinal symptoms are so mild as not to interfere with work. By "poor or failure" is meant those patients having a recurrence of ulcer, persistent ulcer pain, perforation or hemorrhage.

Change in Status: As might be expected, there has been some change in postoperative clinical evaluation among the patients of both groups. Among the postvagotomy patients, during the past year, three moved from "fair" to "good"; one moved from "poor" to "good." None showed regression. Although prior follow-up of our gastrectomy patients has been less complete than for our postvagotomy patients, we know of two cases whose evaluation has moved from "fair" to "good" and one which went from "fair" to "poor."

SUMMARY

1. Forty-nine patients who had vagotomy and 33 who had gastric resection, which constitute all of the cases since the opening of this hospital, have been reviewed and compared.

2. There have been no hospital deaths in either series with the exception of one case moribund from acute hemorrhage, who was explored only as a last resort.

3. Surprisingly enough, the number and severity of complications have been higher following vagotomy than following gastrectomy.

4. Results thus far are somewhat in favor of vagotomy with drainage over subtotal gastrectomy.

The apparent advantage of vagotomy over gastrectomy will be further enhanced when we consider the following three factors: a. Although in our small series there has been no mortality following either procedure in elective cases, the literature shows a definitely greater number of fatalities following gastrectomy than following vagotomy. b. The great majority of patients who have undergone gastrectomy remain gastric cripples, whereas the only limitation in diet, tobacco and alcohol imposed upon our postvagotomy patient has been "everything in moderation." c. In this particular comparative study, 50 per cent of the gastrectomy series were for gastric ulcer which are notorious for their favorable response to any type of surgery.

BIBLIOGRAPHY

1. Dragstedt, L. R., and Owens, F. M., Jr.: Supradiaphragmatic Section of Vagus Nerves in Treatment of Duodenal Ulcer, Proc. Soc. Exper. Biol. & Med. *53:*152-154 (June) 1943.
2. Dragstedt, L. R.: Vagotomy for Gastroduodenal Ulcer, Ann. Surg. *122:*973-989 (Dec.) 1945.
3. Crile, G., Jr.; Jones, T. E., and Davis, J. B.: Surgical Treatment of Duodenal Ulcer—Comparison of Results with and without Vagotomy, Ann. Surg., *130:*31, 1949.
4. Griswold, R. A.: A Rationale for the Surgical Treatment of Duodenal Ulcer, Surg., Gynec. & Obst. *88:*585, 1949.
5. Schoen, A. M., and Griswold, R. A.: The Effect of Vagotomy on Human Gastric Function—A Preliminary Report, Ann. Surg. *126:*655-663 (Nov.) 1947.
6. Hollander, F.: The Insulin Test for the Presence of Intact Nerve Fibers after Vagal Operations for Peptic Ulcer, Gastroenterology, *7:*607-614 (Dec.) 1946.

ABDOMINAL AORTOGRAPHY*

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ABDOMINAL aortography is an additional aid in the roentgen diagnosis of intra-abdominal disease. By the instillation into the aorta of a radiopaque dye, the confirmation or detection of a number of lesions is possible. Some of the more common of these are: Tumors of the kidneys, adrenals or retroperitoneal space, congenital anomalies of the abdominal viscera and aneurysms or occlusions of the aorta or its major branches, especially the renal and splenic arteries.

This procedure has been used rather extensively by our urologic department in their diagnostic work-ups and to a lesser degree by the general and vascular surgical services.

In this paper the technic of abdominal aortography which we have used will be described. Several interesting cases of lesions of the abdominal aorta which were confirmed by aortography will be presented.

TECHNIC

Various technics of abdominal aortography have been described in the literature. The method herein given is simple and relatively safe. We have had no fatalities or serious untoward reactions.

The patient is placed on the x-ray table in the prone position and is anesthetized with intravenous sodium pentothal. The lower thoracic and lumbar regions are prepared and draped. A point is located just below the twelfth rib, at the level of the first lumbar vertebra, approximately 8 to 9 cm. to the left of the midline (fig. 1a). At this point a 6 inch, No. 18 gauge needle with obturator, is introduced at an estimated 45 degree angle, directed slightly cephalad. When the vertebral body is contacted, the point of the needle is allowed to slide past it until resistance is met. When the resistance is overcome, the obturator is removed (fig. 1b). If the needle is in the aorta, there will be a pulsating blood flow from its lumen. A Luer-lock syringe fitted with firm rubber tubing and adapter and filled with 10 c.c. of 80 per cent sterile sodium iodide is connected to the needle. A 14 by 17 inch roentgenogram is made using rapid K. U. B. technic. The film is exposed as the last cubic centimeter of dye is injected.

A normal aortogram with good filling is shown in Figure 2.

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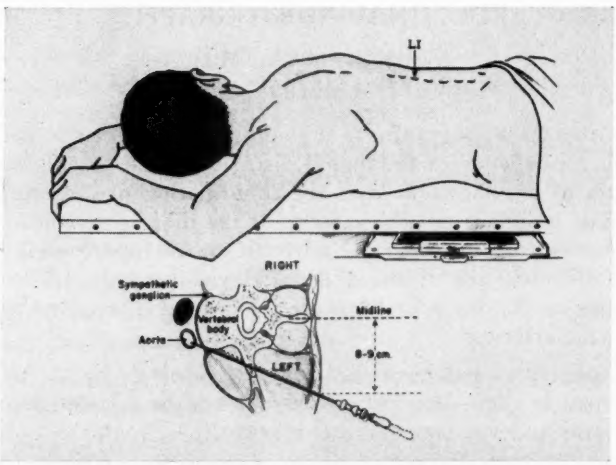

Fig. 1. The technic of abdominal aortography. (a) The position of the patient on the x-ray table. (b) Direction of the needle into the aorta.

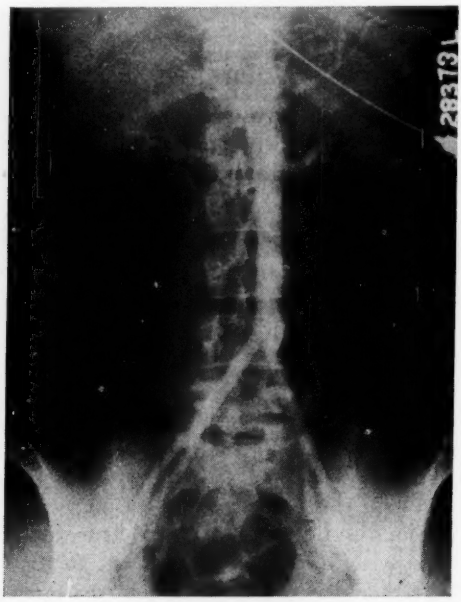

Fig. 2. A normal aortogram with satisfactory filling of the aorta and its major branches.

Case Reports and Discussion

M.C., a 49 year old white female, was first seen in the Emergency Room

of the Louisville General Hospital on the evening of March 18, 1949. Her chief complaint was numbness and paralysis of both lower extremities. Hospitalization was advised, and at the patient's request she was transferred by ambulance to a private hospital.

The history obtained was incomplete as her condition was critical, and there was no competent informant accompanying her.

Fig. 3. Dye is not visualized in the aorta. The visceral arteries are abnormally well filled. The kidneys appear nearly opaque due to extreme filling of the small arteries. The mesenteric arteries are visualized. In the region of the left kidney the finer vessels to the small intestine are so well filled that a relatively opaque appearance is present.

She stated that she had been in good health until approximately 6 p.m., March 14. At this time she had experienced numbness in both lower extremities. This sensation was transitory, lasting only a few seconds. Following this, she was symptom-free. At approximately 9 p.m. that same night she suddenly experienced coldness, numbness and paralysis of her right lower extremity. The paralysis was complete, and she had to be assisted to bed. These symptoms persisted until approximately 7 p.m. the night prior to admission when she suddenly experienced similar symptoms in the left lower extremity.

Her previous history was noncontributory. No history of heart disease could be elicited.

Physical examination revealed a well developed, obese, white female appearing acutely ill. The admission temperature was 98.6°; the pulse was irregular, being approximately 144 per minute, and the blood pressure 108/80. The heart was normal in size. The rhythm was totally irregular. There was a loud systolic murmur at the apex transmitted to the axilla. A systolic mur-

mur at the base, particularly in the right first intercostal space, was transmitted down the right and left sternal border. A thrill at the base could not be demonstrated. The lung fields were clear. The abdomen was not remarkable. Examination of the extremities revealed the lower extremities to be pallid and cool. There was complete loss of sensory and motor function. No peripheral pulsation was detectable in the lower extremities. There was no evidence of gangrene, and both extremities had the same appearance.

Laboratory examination revealed the urine to contain one plus albumin, and on microscopic examination there were 8 to 12 finely granular casts per low power field. The red blood count was 4.09 with 73 per cent hemoglobin. The white blood count was 17,450 with 90 per cent polymorphonuclear leukocytes and 10 per cent lymphocytes.

An electrocardiogram showed auricular fibrillation and an inverted T wave in Lead I.

A diagnosis of saddle thrombus of the aorta complicating rheumatic heart disease with auricular fibrillation was entertained. Because of the long duration of the occlusion it was decided to do an aortogram before embolectomy was attempted. An abdominal aortogram was done at 11 p.m. the night of admission* (fig. 3).

The patient tolerated this procedure poorly. Her condition became much worse, and she was put under an oxygen tent. The following morning there was marked reticulated cyanosis and impending gangrene of both lower extremities. At this time it was felt that although embolectomy would probably be unsuccessful, it would give the only chance for life.

At 10 a.m. on March 19, approximately twelve hours after hospital admission, an aortic embolectomy was done through a retroperitoneal approach. The left iliac artery was opened. A large thrombus which extended approximately 3 to 4 cm. above the bifurcation was milked from the aorta. The thrombus in the right iliac artery was milked around and expressed through the incision in the left iliac artery. The vessel was then closed with everting, interrupted mattress sutures of 000000 silk. Temporary ligatures which had previously been applied were removed, and circulation was reestablished. Pulsation could be seen in the aorta and both iliac vessels. The patient's condition became very poor during the procedure, and she died shortly after the operation was completed.

The aortogram in this case (fig. 3) presents an interesting picture insofar as no dye is visualized in the aorta while the abdominal visceral arteries are abnormally well filled. Two factors probably account for this type of filling. One is probably an error in technic. The film was taken two seconds following the injection of the last cubic centimeter of dye rather than simultaneously with its injection, thus allowing the dye to leave the aorta. The other possibility for the unusual filling, we feel, can be accounted for by the occlusion at the bifurcation, because of which more dye is forced into the branches of the aorta.

The diagnosis of acute aortic occlusion is usually easily made from the history and physical findings. I feel that in this case the anes-

*Aortogram done by Dr. Robert Lich, Jr.

thesia used at the time of aortography and the additional insult of injecting the dye into the aorta were injurious to this patient. We have not tried it on any similar cases and do not feel it is justified.

T. O., a 49 year old white male, was admitted to the Jewish Hospital on May 17, 1949, complaining chiefly of abdominal pain. He stated that he had been in good health until six weeks prior to admission. At that time he had begun to experience a dull aching sensation in his lumbar region and mid-abdomen. The pain became progressively more severe and was most exaggerated in the prone position. During the two weeks prior to admission it would frequently awaken him from his sleep.

Fig. 4. The aorta fills well to the level of the third lumbar vertebra. At this point the column of dye appears to be obstructed.

He was seen by his physician who made a diagnosis of abdominal aneurysm and had him admitted to the hospital.

The systemic, previous and family history were all noncontributory.

Admission physical examination revealed a temperature of 99°, pulse rate of 85 per minute, respiratory rate of 20 per minute. The blood pressure was 125/75.

He was well developed and nourished and did not appear acutely or chronically ill.

Examination was essentially negative except for the abdominal findings. There was a large, well defined smooth mass at the level of the umbilicus. The point of greatest prominence was slightly to the left of the midline. This

mass had a vigorous pulsation synchronous with the heart beat. There was no bruit or thrill present. Both lower extremities were normal, and peripheral pulsations were normal and equal.

Laboratory work was within normal limits. The blood Kahn was negative.

A diagnosis of aneurysm of the abdominal aorta due to arteriosclerosis was made.

*Aortography was done on May 20 to determine the exact location and extent of the aneurysm, as well as the appearance of the aorta proximal to the lesion (fig. 4).

On May 25, under spinal anesthesia supplemented by pentothal, the abdominal aorta was exposed through a retroperitoneal incision. The aneurysm was fusiform in shape and measured approximately 12 cm. in the long axis. The wall was thin and pulsated vigorously. Flakes of calcium could be felt in it. On the anterior surface there were three areas approximately 0.5 cm. in diameter. They were dirty green in color, and the tissue appeared necrotic. These areas appeared to be points of imminent rupture. The aorta proximal and distal to the aneurysm was not remarkable.

The aorta was partially occluded just proximal to the aneurysm with two bands of ¾ inch wide, sixteen-ply polythene cellophane. The aneurysm was then wrapped with four-ply cellophane of the same type. (This case is being reported in detail in a subsequent paper.)

Aortograms in cases where an aortic aneurysm exists may be of great value. In doubtful cases it may confirm the diagnosis. Further, it may definitely help to localize and give the extent of the lesion. The latter is important if surgical therapy is planned. In

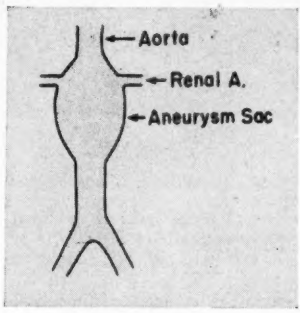

Fig. 5. A diagramatic sketch of an aortogram in a case of abdominal aneurysm. The renal arteries arise from the aorta at the superior pole of the aneurysm.

cases where major tributaries are involved, surgery may be contraindicated, and useless exploration will be avoided. We have had such a case. A diagnosis of aneurysm of the aorta was made from

*Aortogram done by Dr. Robert Lich, Jr.

the history and physical examination. Aortography was done to confirm the diagnosis and further study the lesion. There was excellent filling of the aorta showing a fusiform aneurysm. The renal arteries were seen to arise from the superior pole of the dilatation. This contraindicated surgical attempt at obliterating or resecting the aorta, and the lesion was not explored. Unfortunately, the aortograms on this case were misplaced before being photographed and cannot be shown. A diagramatic sketch is presented (fig. 5).

The aortogram presented in Figure 4 shows the dye filling the aorta to stop abruptly at the proximal end of the aneurysm. This gives the impression that there is an occlusion of the aortic lumen. However, the clinical findings of vigorous pulsation of the aneurysm and palpable pulsation of the femoral arteries bilaterally does not substantiate an occlusion. We feel that this type picture is due to abnormal currents in the aneurysm which probably delay the flow through it. We have not had enough of these cases, as yet, to study this phenomenon but hope to do this in the future with the use of serial films.

The following two cases are of the same nature and will be presented together briefly before discussing the aortograms. These cases are being reported in detail in another publication.

M. E., a 50 year old white male, was first admitted to the Louisville General Hospital on Jan. 8, 1946. At this admission a diagnosis of infarction of the myocardium due to coronary occlusion was made. His course was uneventful, and on March 4 he was discharged as improved. Ten days following discharge he was seen in the Medical Out-patient Dispensary. He failed to return for further check-ups until Jan. 31, 1949, at which time he complained of pain in both legs. He was advised to have further study but did not return to the clinic until Aug. 19, 1949. At this time hospitalization was recommended. On August 22 he was admitted to the Louisville General Hospital. His complaint on admission was pain in both lower extremities. He stated that following his illness in 1946 he began to experience bilateral intermittent claudication. The pain in his lower extremities had become so severe he was forced to quit work. He further stated that his feet were always cold. He had not noticed any color changes of the skin of the extremities, but he thought his legs were smaller and weaker than before.

The past history revealed typhoid fever at the age of 14, syphilis in 1915 with treatment, and coronary heart disease in 1946.

The family history was noncontributory.

He had been smoking approximately two packages of cigarettes a day; his drinking habits were moderate.

History of systems was irrelevant.

Physical examination on admission revealed normal temperature, pulse and respiration. The blood pressure was 120/65. Pulsation of abdominal aorta was absent. While recumbent the lower extremities were markedly pallid and remained so when dependent. No pulsations were palpable in the peripheral

arteries. Oscillometric readings were zero at all levels on both lower extremities. Skin temperatures taken in a warm room revealed an approximate 5° vasoconstrictor gradient.

Admission laboratory work was within normal limits. This included a negative blood Kahn.

Electrocardiogram showed low voltage and a left axis shift.

A diagnosis of chronic occlusion of the terminal aorta was made.

On Sept. 1, 1949 an abdominal aortogram was done (fig. 6). This revealed an obstruction to the flow of dye in the aorta just below the renal arteries.

Fig. 6. There is satisfactory filling of the aorta and its major branches to just below the origin of the renal arteries. At this point the flow is arrested.

This roentgen diagnosis confirmed our clinical findings of occlusion of the abdominal aorta.

On September 8, through a retroperitoneal approach, the terminal aorta and iliac arteries were explored. The aorta below the renal arteries and the iliac arteries were found to be occluded and were resected. A left lumbar sympathectomy was done at the same time. On September 26, a right lumbar sympathetic ganglionectomy was performed.

On October 5, the patient was discharged from the hospital improved.

R. K. F., a 62 year old white male, was admitted to Norton Memorial Infirmary Nov. 1, 1949, because of aching in the left thigh and leg when walking. He stated that he had been in good health until five years ago at which time he had a coronary artery occlusion. He had made a good recovery

from this but had limited his activities. About two years ago he began to experience pain in his left knee when ambulatory. If he continued to walk the pain would radiate superiorly, and he would experience a severe cramplike pain in the upper thigh which would become so severe that he had to stop. He had sought help unsuccessfully from a number of sources. During this period he began to experience more pain with less stimulus. Six weeks prior to admission he began to experience a dull pain in his left calf. Approximately two months ago x-rays of the lumbar spine, and left hip and femur were essentially negative.

Three months prior to admission similar symptoms appeared in the right thigh and knee but to a lesser degree.

Fig. 7. There is satisfactory filling of the aorta and its major branches to the level of the bifurcation. The left common iliac artery is not visualized. Narrowing of the lumen is demonstrated at the bifurcation of the aorta and at the proximal portion of the right iliac artery.

Physical examination revealed the temperature, pulse and respiration to be within normal limits. The blood pressure in the upper extremity was 160/76.

The positive findings were limited to the lower extremities. The color of the extremities was normal. In the recumbent position both feet showed a marked pallor. On dependency the right foot slowly became pink, but the pallor of the skin of the left persisted. There was muscular atrophy of both calves and thighs. This was most noticeable in the right calf. However, there was an anomaly of the right ankle. (The patient stated that this extremity

had always been smaller.) No arterial pulsations including the femoral could be elicited on the left. On the right, the pulsations, although diminished, could be felt.

There was no edema, and the venous tree appeared normal. Perspiration was within normal limits. Skin temperatures were not taken. However, in a warm room of approximately 80°, both feet felt cool, the left being cooler than the right. After walking approximately thirty yards the patient was forced to stop because of severe pain in the left thigh and calf.

Admission laboratory work was not significant. A diagnosis was made of occlusion of the left iliac artery, possibly at the level of the aortic bifurcation.

Abdominal aortography was done on November 8 (fig. 7). This revealed good filling of the aorta and its major branches. At the bifurcation there was narrowing of the aortic lumen, and the proximal end of the right iliac artery. There was no filling of the left common iliac artery. It was occluded at its junction with the abdominal aorta.

This roentgen finding again corroborated our clinical impression, and on November 10, through a retroperitoneal approach, the aorta and iliac vessels were explored. The left common iliac, internal iliac and external iliac arteries were completely occluded. The aortic wall at the bifurcation felt thickened. There was pulsation in the right iliac artery. The left common and proximal segments of the internal and external iliac arteries were resected, and a left lumbar sympathectomy was done.

The patient had an uneventful recovery and was discharged from the hospital Nov. 20, 1949, much improved.

In these two cases of chronic aortic occlusion we feel that aortography was of definite value. Not only did it help to confirm our clinical diagnosis, but also it revealed the exact proximal extension of the thrombosis. Further, in the second case, it has revealed to us early occlusion of the right iliac artery before it was complete. In view of this, we will be able to study better the progress of this relatively rare condition. A sympathectomy is planned on the right, to be followed by aortograms to watch the progress of the disease.

Summary and Conclusions

A technic of abdominal aortography has been presented. The procedure is simple, and very little special equipment is necessary. It is relatively harmless, and in our series we have had no fatalities or untoward reactions.

Five cases have been presented.

In the case of acute aortic occlusion due to a saddle thrombus, we feel that little additional information was obtained by aortography. The procedure in an already desperately ill patient was harmful. In fact, the mere administration of an anesthetic was dangerous. Such cases in nearly all instances are easily recognized, and if seen early enough, aortic embolectomy should be done without loss of time.

Two cases of aneurysm of the abdominal aorta were presented.

One of these was explored and treated, while in the other case, the aortogram revealed that both renal vessels took their origin from the portion of the aorta involved, and the patient was not subjected to surgery which was contraindicated.

Aortograms in suspected abdominal aortic aneurysms are of distinct value. Not only do they corroborate the clinical impression, but they also reveal the exact level of the lesion and in most cases its extent as well. Useless exploration is avoided when major branches, such as the renal arteries, are involved.

In Figure 4 the aneurysm failed to fill. We do not know whether this was a technical error, but are inclined to believe it was due to abnormal currents in the aneurysm. We hope to gain further knowledge of this type of filling defect as more aneurysms are studied with serial x-rays.

In the last two cases presented, we feel that aortography was of decided value. Chronic occlusion of the aortic bifurcation is rare, and although it may be suspected clinically, the exact diagnosis can only be made by aortogram or exploration. Further, the cephalad extension of the thrombosis can be accurately seen. In a case such as the last presented, a partial occlusion can be visualized, and repeated aortography will be of value in studying the progress of the disease.

I feel it is worthwhile to mention in closing that in the two cases of chronic aortic occlusion, no large collaterals to the affected extremities were visualized. However, both of the patients improved following resection of the occluded segments and lumbar sympathectomy. We do not feel that the failure to visualize collateral arterial channels should be a contraindication to the surgical therapy of this disease.

THE LATE SYMPTOMS OF CARCINOMA OF THE PANCREAS*

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CARCINOMA of the ampulla of Vater, carcinoma of the lower end of the common bile duct, and carcinoma of the duodenum are curable by pancreatoduodenal resection. This procedure, so brilliantly conceived and developed, has proved disappointing in its utilization on carcinoma of the pancreas. The patient's preparation is complicated, the operation requires time, the operative mortality while decreasing remains high, the convalescence is lengthy, and the results approximate those following palliative procedures. Carcinoma of the head of the pancreas has not been cured by pancreatoduodenal resection. This unsatisfactory situation exists only because carcinoma of the pancreas in its early, curable stage is a silent lesion.

The onset is insidious and the initial symptoms are easily confused with those which may arise from numerous other causes. Disturbances of digestion, gaseous distention with a sensation of fulness in the abdomen, a decrease in appetite, and constipation are not only not characteristic of any one disease, but when occurring in patients during the fifth and sixth decade do not cause any immediate concern. Only when such symptoms develop suddenly and persist in an individual previously well can their true significance be suspected. The mature symptomatology develops from an extension of the original growth, the position of which determines the symptoms which are produced by an invasion or compression of neighboring structures. Weight loss, asthenia, pain, and jaundice are evidence of this extension; and unfortunately it is their presence, either alone or in combination, which leads to such studies as may be necessary to identify the lesion.

In this series of 102 consecutive operated cases of carcinoma of the pancreas there were 57 men and 45 females. This distribution differs with those of other series in which the ratio is between 2 and 4 to 1. Their ages ranged from 28 to 80. It is worthy of note that this disease is rare in younger patients, there being but eight cases below the age of 50. The average age was 61 years, and this is consistent with the age distribution of other series.

In over 90 per cent of normal anatomic specimens the common bile duct passes through the head of the pancreas for a distance of from 1 to 3 cm. That growths in this region do not obstruct the duct in their operable stage is an unexplained and tragic phenomena.

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It is a well recognized fact that many carcinomas of the head do not produce jaundice prior to operation and that some do not do so prior to death. There were 65 lesions found in this location. Of these 44 were seen after the onset of a constant, progressive jaundice. While a history of mild digestive complaints could be elicited in some cases and definite right upper quadrant pain was present in 5, 32 patients stated that their health had been excellent until the appearance of the painless jaundice. And providing that the sequelae of jaundice had not developed, their general physical condition as was evidenced by the absence of anemia, loss of weight, and asthenia remained relatively unimpaired. That this group was silent is presumptive evidence that pain is initiated only by extension of the growth beyond the boundaries of the gland.

The 21 lesions of the head in which jaundice was not present created one of three clinical pictures. Indigestion, right upper quadrant pain, nausea, some degree of weight loss, and in rare instances vomiting, were represented to some degree in each case. When the cholecystogram showed either no function or stones—cholelithiasis was found in 13 cases of carcinoma of the head—the preoperative impression was that of gallbladder disease. Positive findings after a barium meal—there were 7 cases of extensive invasion into the stomach or duodenum—resulted in a preoperative diagnosis of pyloric obstruction due to either ulcer or malignancy. And the detection of a mass—28 lesions of the head were palpable—made a correct diagnosis possible.

The amount of fat, the condition of the musculature, the depth of the abdominal cavity, the character and physical contour of the liver, and the individual's ability to relax are factors common to every physical examination of the abdomen. If one is searching for an enlarged gallbladder, the probable presence of gallbladder disease is an additional consideration. In the 44 lesions of the head with jaundice the gallbladder could be outlined in 31 patients, a circumstance not only of great assistance in establishing the diagnosis but also of greater solace in planning the operative procedure to be employed. Courvoisier's law proved to be a valuable aid to diagnose in 70 per cent of these cases. The important point is that this law, as is true of others, is reliable only when the gallbladder can be detected.

The 38 patients with growths in the body and tail (four of these were generalized throughout the organ) presented an astounding range of complaints; from "feeling badly," to mild indigestion, to serious indigestion with impaired appetite and associated weight loss, and either with or without pain. The body rests upon the vertebral column and an extension of a growth in this area may involve

the retropancreatic tissues. It is apparently involvement of regional nerve trunks which causes pain. The pain of body lesions, when present, is in general felt in the epigastrium and of tail lesions, when present, in the left hypochondrium; in either case the pain may radiate through the body into the back. This pain varies widely in intensity; it may be constant or may be intermittent with severe exacerbations. When narcotics are required and particularly when pain is present in the back, the presence of a lesion in this region must be disproved. In 29 cases pain was described in either the epigastrium or in the left hypochondrium and in 17 the characteristic back pain was present.

The preoperative study placed these 38 cases into one of three categories. There were 6 patients whose cholecystogram showed either no function or stones; 9 lesions of the head and tail were accompanied by cholelithiasis. These were considered to have gallbladder disease requiring surgery. A palpable mass in 25, 17 being in the body and 8 in the tail, led to a correct evaluation of the pathology. And in 4 cases, the preoperative diagnosis was presumptive, the operative procedure being exploratory in character.

Anorexia, weight loss, and anemia were uncommon in this series. Loss of weight in 24 instances was sufficient to have been noted by the patient prior to examination; and in 14 instances it was pure evidence of a definite pathologic process. The red blood count was below 4,000,000 in but 23 cases and a profound anemia was found in only 9. Nausea and vomiting were limited almost entirely to those cases in which the pylorus and duodenum had been invaded.

The possibilities to be found in roentgen studies of carcinoma of the pancreas have been described many times and the value of these findings in establishing a correct diagnosis has been reported on different occasions. When the first 80 cases in this series were reviewed, it was immediately obvious that such studies had contributed little with the exception of those cases in which the surrounding hollow viscera had been invaded. In the last 22 cases particular attention was devoted to this phase of the study. With the exception of 4 cases of pyloric obstruction, the findings were negative. This was most disappointing in 7 patients, none of whom had either jaundice or a palpable mass and 4 of whom were free from pain. In this series of cases the roentgen studies have made no appreciable contribution.

It is distressing to accept the fact that these patients without exception were operated on in that stage of their disease where either nothing at all could be done or only a palliative procedure adopted. The relative absence of anorexia, weight loss, and anemia suggests

that the growths might not have been too far advanced. In the carcinomas of the head, metastasis to the liver was found in 11, extension to the gallbladder in 3, and invasion of the pylorus and duodenum in 7; in carcinomas of the body and tail metastasis to the liver was found in 5 and invasion of the stomach in 3. An interesting commentary on this disease is that it is not only difficult to diagnose but it is in some instances impossible to identify when the abdomen is open. That a biopsy which fails to include carcinoma cells is unreliable is fully appreciated. During the collection of this series six lesions believed at operation to be malignant were encountered.

Summary

The early, curable malignant lesion of the pancreas is silent. The initial symptoms are mild, indefinite, and not disturbing. Jaundice, pain, weight loss, and anemia, are all evidence of the growth's extension beyond the gland. Even when a presumptive diagnosis of such a lesion is made on a basis of the history in the absence of any demonstrable finding, the lesion proves to be inoperable. Magnificent advances have been made in the surgical management of pancreatic disease. Until similar advances can be made in diagnosis, the fruits to be enjoyed by pancreatoduodenectomy cannot be touched.

THE INCIDENCE OF PEPTIC ULCER FOLLOWING SYMPATHECTOMY FOR HYPERTENSION

Presentation of a Case*

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SURGERY of the autonomic nervous system for systemic disease is not a new concept, and during the past ten years such surgery has taken tremendous strides. Claude Bernard in 1852 was the first to bring attention to the sympathetic nerve supply of the cardiovascular system and its importance in the maintenance of the "status quo" of the individual. Jaboulay in 1899, and Leriche in 1913, performed the first periarterial sympathectomies; Leriche in 1926 introduced the extraperitoneal approach to the sympathetic chain.[1] Beginning in 1927, and on, Piere, Craig, Adson and Brown, Peet and Smithwick, and others, made important contributions to sympathetic surgery for hypertension.[1-4]

Surgery of the autonomic nervous system for disorders of the gastrointestinal tract also is not a new concept. Vagotomy for peptic ulcer was performed by Stierlin in 1920, Bircher in 1921, Schiassi in 1925, Barrow and Curtis in 1937, and Berg in 1938. However, the real impetus for such surgery was supplied by Dragstedt and Owens in 1943.[3]

Our purpose in this discussion is to present a case of duodenal ulcer making its first frank appearance following sympathectomy for hypertension. Dragstedt cautioned when he said, "There is some evidence that sympathectomy in these patients (hypertension and ulcer) may cause a rapid progression in the ulcer, but at the same time produce relief of pain so that a painless perforation may occur.[5] Wangensteen also warned, "Sympathectomy definitely abets the ulcer diathesis in dogs and we might reasonably assume that some patients who have undergone sympathectomy for hypertension may develop ulcer."[6] Our purpose is to present such a case.

The physiology of the autonomic nervous system, as applied to the gastrointestinal and cardiovascular systems, is not a field of undisputed fact; in reality, it is difficult to find two authorities on the subject who are in anything like agreement.[7-15] It is generally felt that a certain balance exists between the sympathetic and the parasympathetic systems. Thus, the idea of autonomic imbalance lead-

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ing to visceral dysfunction, as expressed by Eppinger and Hess in 1909, seems to have some basis.[16-18]

Briefly, it is generally accepted that stimulation of the vagi causes an increase in gastric secretion which is high in acid and pepsin, and a decrease in emptying time of the stomach.[16]

The effects of stimulation of the sympathetics in man are not so well accepted, but most investigators will agree that such stimulation leads to a secretion high in alkaline mucus, and an increase in gastric emptying time.[8,10,15,16] Thus, it has been postulated that section of the sympathetics in man will lead to visceral dysfunction, manifesting itself in a more acidic gastric secretion, and a marked decrease in emptying time, due to the relative increase of vagal activity. Another very important function of the sympathetics, referred to before, is that of mediation of intra-abdominal sensation.[5,11,18,19] The sympathetic preganglionic innervation of the abdominal viscera arises from cord section T4-L1, and, as the splanchnics, continue to the celiac plexus whence the postganglionic fibers arise.

The theories behind sympathectomy for hypertension can briefly be summarized in two basic groups. First, there are those who believe that the important end-point is to increase the peripheral vascular bed and thus form a so-called pooling of blood, at the same time reducing peripheral vascular resistance. These are the men who do the more complete procedures such as the supradiaphragmatic and infradiaphragmatic procedure of Smithwick.[3] The other school of thought is that the important factor is to alleviate the relative renal ischemia that is thought to exist and thus reduce the production of angiotonin. These men follow the supradiaphragmatic procedure of Peet.[2]

However, regardless of the type of sympathectomy done for hypertension, those nerves supplying the abdominal viscera are the ones sectioned.

Presentation of a Case

The case that we are presenting is that of a 36 year old colored male who was first seen at the Louisville General Hospital in April, 1945, with chief complaints of vertigo, severe headache, shortness of breath on exertion and nocturia. His blood pressure was 220/134, the electrocardiogram revealed left axis deviation, and urinalysis showed specific gravity of 1.012 with 2 plus albumin. An incidental finding in the past history at that time was a vague epigastric distress precipitated by certain foods and relieved by soda. A diagnosis of essential hypertension was made and the patient was placed on a reducing diet and sedatives.

In January, 1946, the patient's blood pressure was found to be 220/160,

and his eyegrounds showing changes consistent with malignant hypertension. Symptomatically the patient had improved, even though his diastolic pressure was up 26 mm. over the previous year. In August of the same year the patient was admitted to the medical ward with aggravated symptoms, including severe nosebleed. On the ward his pressure fell to 200/100. Symptomatically the patient improved—his nonprotein nitrogen was 46, urea clearance 77 per cent, the Mosenthal showing relative inability to concentrate. A diagnosis of arteriolar nephrosclerosis was made.

In the next month the patient was readmitted for studies prior to surgery. His fundi showed many old exudates. His urea clearance was 30 per cent, but at the same time, nonprotein nitrogen was lower. Retrograde pyelography was considered normal. In further studies, under light pentothal anesthesia, the blood pressure dropped 60 mm. systolic and 16 mm. diastolic; when the anesthesia was carried to deeper levels on a subsequent day, the pressure dropped to 120/70. This patient was considered a suitable candidate for sympathectomy and on Sept. 26, 1946, he was subjected to a left supra- and infradiaphragmatic sympathectomy, splanchnicectomy, and subtotal excision of the celiac plexus. The sympathetic chain from T8-L2 was removed. Postoperatively, the patient's pressure stabilized around 180/110.

Two months later the patient was admitted for completion of the procedure. In the interim he had continued to have as many symptoms as prior to surgery. His fundi still showed old exudates, silver wire appearance and nicking. The Mosenthal showed greater concentrating ability, but the night volume was recorded as 690 c.c. Preoperatively the pressure was 190/110. At operation the same procedure as before was carried out on the right. Postoperatively he was bothered by postural hypotension—his pressure finally stabilizing at about 150/100 prone and 130/85 standing.

During the following months, until 1948, the patient experienced complete relief of symptoms and apparent reversion of fundic changes.

In April, 1948, he was admitted to the medical ward with chief complaint of extreme weakness, palpitation, black stools and epistaxis. He complained of no abdominal distress. His red blood count was 1,870,000 and nonprotein nitrogen was 38. After the acute episode was over, barium studies of stomach and duodenum showed a spastic and distorted duodenal cap and gastric analysis revealed 40 units of free acid. A diagnosis of bleeding peptic ulcer was made. He was discharged on an ulcer regimen and repeated x-ray studies made during the remainder of the year showed persistent duodenal cap changes with 20 per cent five hour retention.

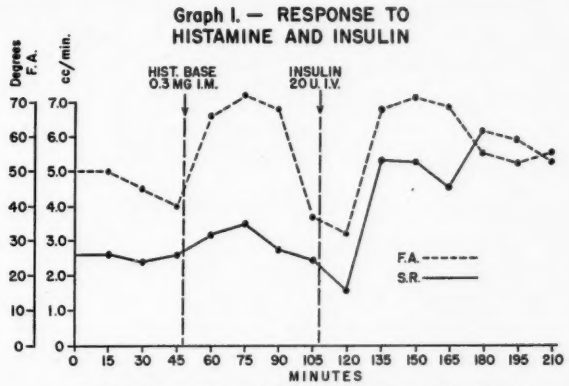
In January of this year (1949) he was again admitted with complaints as before, i.e., tarry stools, weakness and hematemesis. Again he had complete absence of abdominal complaints. His blood pressure was 160/110. Red blood cells 2,710,000, with hemoglobin 8 Gm. During this period studies were again performed, and the impression again was bleeding duodenal ulcer. Because of these recurrent and moderately severe bleeding episodes, with complete absence of abdominal pain, it was felt that the only safe procedure would be to treat the ulcer surgically as a prophylactic measure against repeated attacks of bleeding. From the patient's response to histamine and insulin, and with the added information of 20 per cent gastric retention at the end of five hours, it was decided the patient would benefit from a vagotomy and gastroenterostomy. The choice of operation was made according to the rationale of Griswold.[17]

At operation a scar was found on the anterior surface of the duodenum. A subdiaphragmatic vagotomy and gastroenterostomy were performed, the postoperative course being uneventful.

The patient has been seen several times since the last operation. He has continued symptom free and in good health. His weight is 165 and his blood pressure is 160/120. The most recent gastrointestinal series, reported in October, revealed a rapidly emptying stomach with no abnormalities other than the gastroenterostomy created in the operating room.

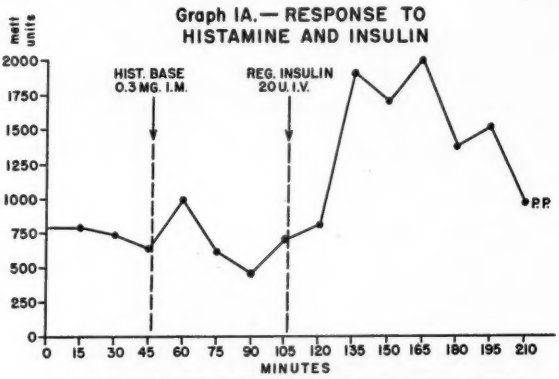
Presentation of Data

Prior to gastric surgery, because of the interest in this problem, i.e., that of peptic ulcer following sympathectomy, and stimulated by the reports of Blegen, et al[18] and Weeks, et al,[19] this patient's gastric function in response to certain drugs was studied extensively using the dilution-indicator method of gastric analysis.[20] Since the completion of these studies, a report of 13 cases of peptic ulcer following splanchnicectomy have been presented by Mason, et al.[21]



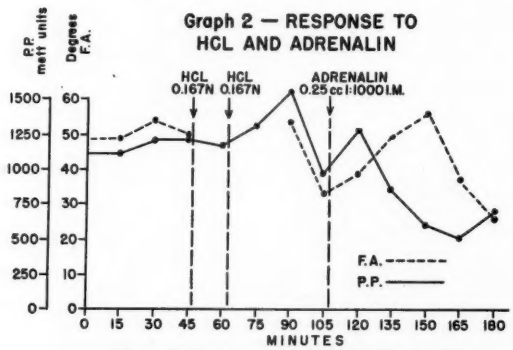
F.A. is degrees of free acid. S.R. is secretory rate as cc/min. The first three periods represent control periods.

Under fasting conditions the patient's response to histamine and insulin were studied. (Graphs 1 and 1a.) The residiuum was 115 c.c. with 50 units of free acid. The high residiuum can probably be explained on the basis of partial pyloric obstruction due to the duodenal ulcer. The basal secretory rates, free acid and peptic power were as shown in the first three control periods. All of these values are in keeping with those of patients having a peptic ulcer. Following the injection of histamine the responses usually seen following this drug were elicited, i.e., secretory rate and free acidity were increased whereas peptic power was decreased as it usually is following the injection of histamine. In response to insulin, the patient's blood sugar fell to 20 mg./100 c.c. and the secretory rate, free



P.P. represents peptic power expressed as mett units. This graph depicts studies done simultaneously with Graph I.

acidity and peptic power were all increased. Due to the preponderance of the response to insulin hypoglycemia, a vagotomy was thought the surgical procedure of choice, to abolish the cephalic phase of gastric secretion.[17]



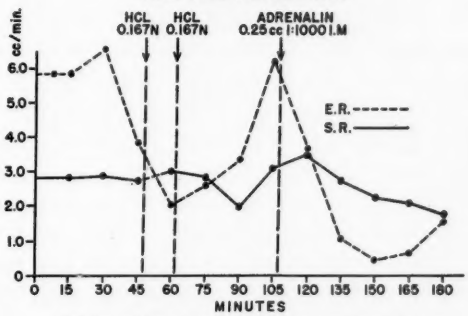
F.A. is degree of free acid. P.P. is peptic power as mett units. The first three periods represent control periods.

The patient's response to adrenalin also was studied before vagotomy and at the same time the patient was subjected to the HC1. "acid test." (Graph 2.) Control periods are represented by the first three periods. Following the control periods 0.167 N HC1 was injected into the stomach to see if the patient would experience pain with its injection, as most peptic ulcer patients do. The results were those to be expected; the patient experienced no discomfort since the sympathetic innervation to the stomach had been removed.

Following the intramuscular injection of adrenalin the patient's

blood pressure was recorded at 255/160, the patient experiencing frontal headache. As shown (Graph 2) peptic power was decreased following a temporary initial rise, while free acidity increased for 45 minutes, before falling to a low value. This finding of increased acidity after administration of adrenalin was repeated in this patient on another occasion and also in another patient studied in a similar manner. The increase may be due to the decreased alkaline secretion as indicated by a decrease in peptic power.

Graph 3 — RESPONSE TO HCL AND ADRENALIN



E.R. is emptying rate in cc/min. S.R. is secretory rate in cc/min. The first three periods represent control periods.

Secretory and emptying rate were both decreased following adrenalin (Graph 3). The interesting finding here is the marked decrease in emptying rate. From this it might be postulated that following sympathectomy there will be a marked increase of emptying rate resulting from removal of the motor inhibitory influence of the sympathetics. To those who believe in the "jet theory" of impingement of acid gastric contents on the duodenum in the etiology of duodenal ulcer, this fact poses an interesting argument. One unfortunate aspect of this particular series of studies was that our estimations of gastric mucus were unsatisfactory—this study would have been most helpful, in that one of the theories concerning the development of peptic ulcer following sympathectomy is a decrease of alkaline secretion following such a procedure.

The patient's response to tetraethylammonium chloride, atropine sulfate and other agents was also studied, but are not included since they add nothing to this present discussion.

Discussion

Painless episodes of hematemesis, tarry stools, weakness, and anemia occurring for the first time two years after a bilateral thora-

columbar sympathectomy for essential hypertension suggested the presence of a bleeding peptic ulcer. Although barium studies of the upper gastrointestinal tract, which were made after bleeding had stopped, failed to reveal an ulcer crater, the presence of a spastic and distorted duodenal cap, and of 20 per cent gastric retention after five hours, favored the diagnosis of duodenal ulcer.

The second episode of similar painless symptoms occurring nine months later was again diagnosed as bleeding duodenal ulcer. A bleeding ulcer which recurs after nine months of prophylactic medical therapy and which is not preceded or accompanied by abdominal pain or any other symptom usually associated with active ulcer, is indication for definitive surgery as a preventive measure against further hemorrhage.

Gastric function studies, made prior to the anticipated surgery for ulcer prophylaxis, showed that the basal acidity and rates of gastric secretion and emptying were increased above normal. The studies suggested that the increased emptying rate was due to the absence of sympathetic inhibition which permitted the unopposed vagal activity to maintain rapid emptying. The studies also showed that the secretion of hydrochloric acid and pepsin as well as emptying rate were all stimulated more by vagal influence than by the injection of histamine, i.e., gastric function was increased more by nervous influence (parasympathetic influence) than by a known potent humoral stimulation of the stomach.

A consideration of the results of the gastric function studies, and the fact that the vagal influence on the stomach was unopposed by the antagonistic sympathetics, led to the decision that vagotomy rather than partial gastric resection should be the operation of choice in this instance. The presence of gastric retention was the indication for combining a low gastroenterostomy with the vagotomy.[17] Accordingly, the combined procedure was performed. The patient has been followed up at regular intervals. He has had no recurrence of bleeding and has remained symptom-free. Barium studies of the stomach and upper intestinal tract reveal a well functioning stoma, a rapidly emptying stomach, and no abnormalities in the stomach, stoma, duodenum and jejunum.

Psychological studies including the Rorschach, Bender-Gestalt, Cornell Medical Index, and various brief progressive tests by the Department of Psychiatry, indicate a patient with strong anxiety and feelings of personal inadequacies conflicting with intense intellectual striving and stubborn resistiveness. Some evidence of hypochondriacal trends are also present. Marked and poorly concealed hostility existing in a personality with considerable effeminacy are also manifested. In general, the psychological studies show person-

ality rigidity, lack of emotional control, and anxiety with inner tension.

The patient has the wherewithal of a psychosomatic personality; that is, inadequacies, hypochondriasis, effeminacy, rigidity of capacity for development of personality relationships, marked anxiety with inner tension, strong but poorly concealed and poorly managed hostility, and marked ambivalence in expression. It is supposed that these findings have previously manifested themselves in the development of the above-mentioned somatic disabilities and that their continuance at the present unresolved level makes this individual susceptible to further complex organic pathology.

It is an interesting speculation that the patient's hypertension was an outlet for the abnormal psychiatric factor noted above. When this outlet was closed off by sympathectomy, it may be that the necessity for another outlet manifested itself in the development of a peptic ulcer.

SUMMARY

1. A case of bleeding duodenal ulcer making its first appearance two years after thoracolumbar sympathectomy for hypertension has been presented.

2. Gastric studies showed that this ulcer was of the vagal "cephalic" type.

3. Vagotomy and gastroenterostomy were performed.

4. Was this ulcer the result of sympathetic-parasympathetic imbalance? If so, was this imbalance due to the sympathectomy or was it a psychosomatic manifestation of his emotional tension?

REFERENCES

1. Goetz, R. H.: The Surgical Physiology of the Sympathetic Nervous System with Special Reference to Cardiovascular Disorders, Surg., Gynec. & Obst. *87:*417-439, 1948.
2. Peet, M. M.: Results of Bilateral Supradiaphragmatic Splanchnicectomy for Arterial Hypertension, New England J. Med. *236:*270-277 (Feb. 20) 1947.
3. Smithwick, R. H.: Surgery of the Autonomic Nervous System, New England J. Med. *236:*662-669 (May 1) 1947.
4. Heinbecker, P.: Factors Limiting Surgery for Essential Hypertension, Ann. Surg. *126:*535-544, 1947.
5. Dragstedt, L. R.; Woodward, E. R.; Harper, P. V., and Storer, E. H.: Mechanism of the Relief of Ulcer Distress by Gastric Vagotomy, Gastroenterology, *10:*200-204 (Feb.) 1948.
6. Dragstedt, L. R.; Harper, P. V.; Tovee, E. B., and Woodward, E. R.: Section of the Vagus Nerves to the Stomach in the Treatment of Peptic Ulcer, Ann. Surg. *126:*687-708 (Nov.) 1947.
7. Hollander, F.: Laboratory Procedures in the Study of Vagotomy, Gastroenterology *11:*419-425, 1948.

8. Babkin, B. P.: Secretory Mechanism of the Digestive Glands, Oa 78, New York: Paul B. Hoeber, Inc., 1944.
9. Wolf, S., and Wolff, H. G.: Human Gastric Function, New York: Oxford University Press, 1943.
10. Alvarez, W. C.: The Mechanism of the Digestive Tract, New York: Paul B. Hoeber, Inc., 1928.
11. Alvarez, W. C.: Pathways for Pain Arising in the Stomach, Gastroenterology *8:*793-794, 1947.
12. Collins, E. N.; Crile, G., and Davis, J. B.: Follow-up of Vagotomy Plus Gastroenterostomy or Pyloroplasty for Ulcer, Gastroenterology *11:*453-456, 1948.
13. Dragstedt, L. R., and Camp, E. H.: Follow-up of Gastric Vagotomy Alone in the Treatment of Peptic Ulcer, Gastroenterology *11:*460-465, 1948.
14. Thomas, J. E., and Komarov, S. A.: Physiological Aspects of Vagotomy, Gastroenterology *11:*413-418, 1948.
15. Dragstedt, L. R.: Section of the Vagus Nerves to the Stomach in the Treatment of Peptic Ulcer, Surg., Gynec. & Obst. *83:*547-549, 1946.
16. Kuntz, Albert: Autonomic Nervous System, 3rd Ed., Philadelphia: Lea & Ferbiger, 1945.
17. Griswold, R. A.: A Rationale for the Surgical Treatment of Duodenal Ulcer, Surg., Gynec. & Obst. *88:*585-591, 1949.
18. Blegen, H. M., and Kintner, A. R.: Aggravation of a Gastric Ulcer Following Dorsolumbar Sympathectomy, J.A.M.A. *133:*1207-1208 (April 19) 1947.
19. Weeks, C.; Ryan, B. J., and VanHoy, J. M.: Two Deaths Associated with Supradiaphragmatic Vagotomy, J.A.M.A. *132:*988-990 (Dec. 21) 1946.
20. Schoen, A. M., and Knoefel, P. K.: The Measurement of Human Gastric Function, J. Lab. & Clin. Med. *32:*345-346 (March) 1947.
21. Mason, S. C., and Pollard, H. M.: Peptic Ulcer Following Splanchnicectomy—A Report of Thirteen Cases, Surg., Gynec. & Obst. *89:*271-283, 1949.

FRONTAL LOBOTOMY FOR THE RELIEF OF INTRACTABLE PAIN

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FROM the earliest days of man pain and its relief has been a major problem. Since pain is only a symptom of a disease process, relief is usually accomplished merely by proper diagnosis and treatment of the primary disorder. In some instances, however, the disease may be such that a cure, and thereby relief of the pain, is beyond hope. It is in these cases, therefore, that the treatment of the pain itself becomes of prime importance. Surgery performed purely for the relief of pain generally implies a destructive operation upon the nervous system. Such operations are: division of a peripheral nerve, rhizotomy, sympathectomy, cordotomy, medullary tractotomy or a combination of these methods. These standard procedures have proved their worth time and again to the neurologic surgeon as a means of relieving suffering when life would otherwise be intolerable. In some instances, however, these operations are not successful in relieving certain painful states, as, for example, painful phantom limbs and neoplasms which involve areas of the body that cannot be easily denervated, as the head and neck, upper extremity and upper chest or a combination of these regions. Recently frontal lobotomy has been added to the armamentarium of the neurologic surgeon as a means of relieving intractable pain, when all other methods have failed or when it is obvious that any of the standard operations would be only temporarily successful. Further consideration of frontal lobotomy as a method of relieving intractable pain is the subject of this paper.

HISTORICAL CONSIDERATIONS

Frontal lobotomy as it is known today was developed by Egas Moniz[10] who in 1936 published a report of 20 cases in which the operation had been done for mental disorders. It is of interest to note that Moniz was encouraged to proceed with his operation following a report by Fulton on the behavioral changes observed in chimpanzees following bilateral removal of the frontal areas.[7] In America the work of Moniz was adopted by Freeman and Watts[2] who also reported in the year 1936 a case of frontal lobotomy performed for psychosis. During the early years of their work with prefrontal lobotomy, Watts and Freeman realized that patients who complained of pain before surgery for their psychosis no longer

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complained of pain in the postoperative period. In 1946 they reported a case upon whom frontal lobotomy was performed purely to relieve intractable pain.[3] In the same year Poppen[13] reported a similar case. The procedure in both of these instances was the conventional bilateral procedure which is a rather complete division of the fiber tracts in the frontal lobe rostral to the anterior horn of the lateral ventricle. The operation as described by Watts and Freeman was a blind procedure performed from a lateral approach.[4] Lyerly[8] described a method to perform the lobotomy by an open operation. A different approach to the study of frontal lobe isolation in the treatment of psychosis was instituted by Penfield.[11] This operation, called gyrectomy, was devised to study the changes in psychotic patients after removal of selected portions of frontal lobe cortex. Pool[12] has carried out similar operations in the human and refers to the procedure as topectomy. Scoville[17] with the same motives for research has devised an operation referred to as "cortical undercutting" by which individual portions of the frontal cortex have been detached from the remainder of the frontal lobes. The latest variation of frontal lobotomy was made in 1948 by Scarff,[15] who reported three cases of unilateral lobotomy which had been successful for the relief of intractable pain.

Anatomic and Physiologic Considerations

The portion of the anatomic frontal lobe which is detached or isolated in frontal lobotomy lies rostral to the motor and extrapyramidal cortex. For many years this portion of the brain has been known as a "silent area." Modern physiologic methods have proved in experimental animals, however, that distinct functions can be demonstrated in several separate portions of the frontal cortex rostral to the motor areas. Frontal lobotomy has opened the way for new discoveries in the function of the frontal areas in man.

It is generally assumed that the success of frontal lobotomy stems from the destruction of the thalamocortical and/or corticothalamic fiber tracts. By study of the human brain following frontal lobotomy Freeman and Watts[5,6] and Meyer, Beck and McLardy[9] have demonstrated connections between the thalamus and every portion of the frontal cortex. The studies of these men have demonstrated loss of ganglion cells and gliosis chiefly in the dorsomedial nucleus following lobotomy. Portions of the medial surface of the hemisphere, in particular the cingulum, project to the anterior nucleus which also shows degeneration after most lobotomies. Strangely enough, little, if any, degeneration occurs in the frontal lobe cortex,[6,9] and this possibly accounts for the fact that within a few

months postoperatively only a few patients show any significant changes in the electroencephalogram as compared with preoperative tracings.

It is now clear that the autonomic system is represented in the frontal areas. The early elevation of temperature, slow pulse and lowering of blood pressure are the only changes that follow lobotomy which can be attributed to temporary disruption of autonomic function. Within a few days after surgery these changes return to normal. There may be permanent changes, however, in the manner in which the autonomic system responds to stimulation.[14]

Wilson and Warland[1] report the development of epileptic seizures in 3.3 per cent of one thousand operations performed for mental disorder. Pool[12] reports the development of convulsions in 10 per cent of 85 cases following topectomy.

Clinical Considerations

Bilateral frontal lobotomy is followed immediately in the postoperative period, in most cases, by drowsiness, confusion and sometimes incontinence. These symptoms, as a rule, clear up within a few days or within two to three weeks at the most. The late effects are frequently a blunting of the intellect and personality. Changes in personality are more common and disturbing than changes in intellect. There also may be loss of initiative and defects in the ability to retain experiences. While such changes may be slight or even undetected in certain cases, nevertheless, some changes in this sphere are so likely that frontal lobotomy of the conventional or standard type can be advised in only a few instances for relief of pain. These undesirable side effects are not severe enough to contraindicate the procedure as a valuable adjunct in the treatment of advanced or inoperable carcinoma and at times it may be indicated in noncancerous conditions such as morphine addiction, tabetic pains or painful phantom limb. It was to avoid changes in personality and intellect that Scarff[15] used a unilateral procedure. His reasoning for the unilateral operation was that since a bilateral operation produced changes in memory and personality and since it was generally believed that a unilateral frontal lobectomy did not cause any changes in personality and intellect, perhaps a unilateral lobotomy would be successful in relieving pain and at the same time cause none of the undesirable side effects.

The author originally performed prefrontal lobotomy for psychotic patients by the method described by Freeman and Watts[4] (fig. 1). Later the method described by Lyerly[8] was adopted (fig. 2). This operation was more rational from a surgical point of view

since it was an open operation, thereby providing control of hemorrhage and more assurance that the proper fiber tracts had been sectioned. A trephine opening made near the midline is placed so that the coronal suture crosses the posterior portion of the button of

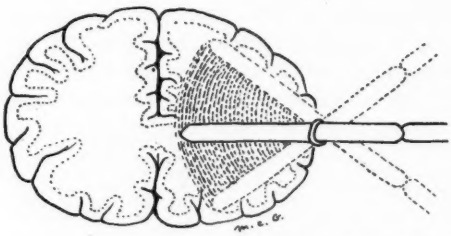

Fig. 1. Schematic drawing of a coronal section of the frontal lobes at the level of lobotomy incisions. The extent of the incision made in one hemisphere by the Watt's technic (lateral approach) is shown.

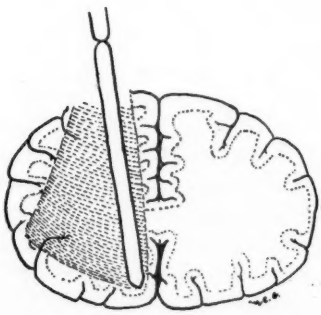

Fig. 2. Schematic drawing of a coronal section of the frontal lobes at the level of lobotomy incisions. The extent of the incision in one hemisphere made by the Lyerly technic (open method) is shown.

bone which is removed. After opening the dura the anterior horn of the lateral ventricle is identified with a ventricular needle. An incision is then made in the exposed cortex large enough to admit brain spatulae for retraction. The fiber tracts are then divided under direct vision until cortical tissue is identified laterally, medially and inferiorly. The following 6 cases have been operated on using this technic—3 cases with the unilateral operation and 3 with the bilateral operation. Both the advantages and disadvantages of frontal lobotomy are clearly demonstrated in these cases.

CASE 1. A 43 year old male was admitted to the service complaining of pain in the right foot. He related that three years previously a heavy steel bar fell on his foot but did not produce any fractures. Because of persistent, incapacitating pain, a surgeon divided the cutaneous nerves to his foot but there was no relief of pain. A cordotomy was then performed which also failed to relieve his pain. The patient also gave a history of treatment for central nervous system lues four years prior to the onset of his present illness. After admission sympathetic blocks were performed without any relief of the patient's pain. He was told of prefrontal lobotomy and elected to have this procedure since he felt he could not go on with his pain. A unilateral operation was performed, the right frontal lobe being used. There was marked relief of his leg pain in the first few weeks postoperatively but within three months the patient stated his pain was as severe as it had been at any time. When seen fifteen months later, he continued to complain of the same severe pain although he was not losing any time from his regular work. His wife, however, states that he complains far less frequently than he did prior to the operation. She has seen minor changes in his disposition. The patient volunteered that he is forgetful for insignificant or unimportant things but had normal ability to retain in his memory those things which would normally be considered of reasonable importance.

Comment: In spite of the fact that he does not admit to any improvement, this patient is working regularly and his wife considers the operation partly successful in that he gets along better in his work and at home. It is probable that his pain from the beginning was of psychic origin and it is quite likely that pain of this type is not benefited to any appreciable extent by lobotomy or at least by a unilateral procedure. Minor but definite changes of personality and memory occurred.

CASE 2. A 72 year old male was admitted to the hospital with an inoperable carcinoma of the larynx which had been present for twelve months prior to admission and for which he had had a tracheotomy. He was suffering with a severe, throbbing, pulsating pain in the right side of his face and neck which was not relieved by narcotics. The patient was in a severe debilitated state, and it was expected that he would live but a short time, but to relieve his suffering during this time, prefrontal lobotomy was advised and accepted by the patient and his family. A right prefrontal lobotomy was performed with a complete section of all the white matter rostral to the tip of the anterior horn of the right lateral ventricle. The patient had complete relief of his pain and no longer required any medication until his death three weeks later. He was confused for a few days but after this he seemed his normal self with the exception of minor changes in his personality which were noticed by his family. The only significant change was that the patient became less tidy in his personal appearance than he had been previously.

Comment: The patient did not live long enough to provide a conclusive study of the effectiveness of unilateral frontal lobotomy to relieve pain. It demonstrated, however, the value of frontal lobotomy to provide physical and mental peace to the patient in his

terminal days. Again minor changes in personality were evident with a unilateral frontal lobe lesion.

CASE 3. A 29 year old female was admitted with a history of severe headaches which were localized to the right side of the head. She stated that the headache began ten years previously during nurse's training. In addition to all known medication commonly employed for headache, treatments had consisted of encephalograms, division of the superficial temporal artery and supraorbital nerve avulsion. The headaches came in severe bouts lasting for a few days to weeks and eventually narcotics were required for relief and this in turn had led to addiction. The patient had been hospitalized on one occasion shortly before this admission for an acute psychosis. After due consideration of her future and in view of the successful relief of narcotic addiction reported by Scarff, a unilateral prefrontal lobotomy was advised for the relief of her pain and the control of her addiction. The conventional (radical) prefrontal lobotomy was performed on the right side by the Lyerly technic. Eighteen months have elapsed since surgery. The patient has continued to complain of some headache but there has been considerable relief. Medication, other than an occasional aspirin, is not required. According to her husband, the patient is more talkative than she was previous to the operation. She has less initiative. She becomes emotionally upset somewhat easier but her memory and intellectual capacities in every other way are felt to be normal.

Comment: As in Case 1 there has been some improvement as judged by the patient and her family but again it clearly shows that a unilateral lobotomy is not completely effective for relieving pain. The changes in personality have been minimal and have not interfered with her ability to carry on in her home. Enough time has elapsed since surgery to be certain that the personality changes, which occurred following a unilateral operation, are permanent.

CASE 4. A 37 year old male was hospitalized because of severe nausea and vomiting and attacks of agonizing lightning pains. There was a history of a luetic infection occurring at the age of 16 which under intensive treatment was thought to be cured. In 1943, however, he began to have attacks of nausea and vomiting which at the time were thought to be due to excessive drinking. In 1944 he was found to have a positive spinal fluid serology. In spite of intensive treatment, the patient continued to have episodes of severe nausea and vomiting associated with lightning pains in his extremities. In an effort to relieve his lightning pains, he had received an injection of some material in the vicinity of his spinal column which resulted in an immediate complete and permanent paralysis and anesthesia of the right lower extremity. At the time of admission the patient was in a deplorable general physical state from weeks of vomiting. He was depressed over the vomiting and the continual shooting pains in his lower extremities. Frontal lobotomy was elected and the bilateral procedure was performed with immediate relief of all tabetic pain. However, he continued to have some nausea and vomiting for the following three weeks, when these symptoms also disappeared. The patient gained weight in the succeeding months, and became a very cheerful, pleasant individual. Since his operation he has started a new business which has been successful. His only disability was the anesthetic and paralyzed leg which caused him a great deal of trouble due to the frequent development of pres-

sure sores. Exactly one year postoperatively he developed a brief episode of lightning pains which were completely relieved following one dose of demerol. The attacks of nausea have occurred in mild fashion a few times but have not been disabling. The only frontal lobe changes that have been detected are minor changes in personality. He is at times uninhibited and somewhat tactless.

Comment: There was a dramatic relief of pain which has been complete except for the brief recurrence one year after the operation. The greater success of the bilateral procedure as compared with the two other cases which had the unilateral operation was quite impressive. There has been no return to narcotics or heavy drinking. To his family and his family doctor, a friend of many years, he has not changed intellectually.

CASE 5. A 49 year old female developed pain and swelling of her right lower extremity. A mass was found in the left lower quadrant of her abdomen which could not be removed at operation. A diagnosis was made of retroperitoneal sarcoma and x-ray therapy was given. Severe right leg pain developed. A frontal lobotomy was advised by her physician and she was admitted to the hospital and the bilateral procedure by the Lyerly technic was performed on Dec. 3, 1948. The patient had complete relief of her pain and no longer required any medication. However, during her hospital stay, she was facetious and at all times confused; her memory was disturbed and she was incontinent. These prefrontal changes did not improve materially before her death three weeks later.

Comment: This case demonstrates the immediate relief of all pain and also the unwanted side effects. The family considered that the relief of suffering which the patient gained justified the operation even though she lived for only three more weeks.

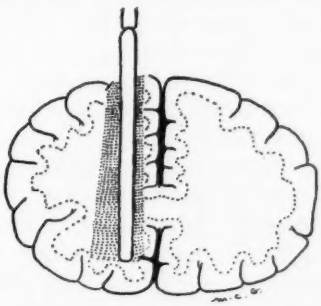

Fig. 3. Schematic drawing showing the type of incision made in Case 6 in which only the medial fibers were incised under direct vision.

CASE 6. A white male, 42 years of age, was seen in consultation because of severe pain after an operation for adenocarcinoma of the rectum at which time the carcinoma was found to be invading all the pelvic structures. In addition, the patient was moderately despondent and anxious over the fact that

he had an incurable cancer. On 19 Jan. 1949, a bilateral prefrontal lobotomy performed by the Lyerly method was carried out but the technic was changed to a less destructive operation. Only the medial fibers in each hemisphere were divided (fig. 3). After he recovered from his anesthetic the patient no longer complained of pain. Ten days later his only abnormality was very slight confusion and difficulty in getting a normal night's sleep. Within another week the patient was free of his confusion. He had been relieved entirely of his anxiety and worry. He did not appear euphoric but was confident of the future. He was a school teacher and felt he could return to his work. Instead he entered the second semester at a college in his home town and finished the term with a B average in his work. When asked, he stated that he had pain but also stated that it did not trouble or worry him. A brother, who is a physician, was the only member of the patient's family, including his wife, who saw any significant change in his personality. This brother noticed that in relating past events the patient occasionally did not adhere to the facts. When another brother died, the patient was less concerned than would have been expected. He walked approximately two miles daily until a few days before his death. Following metastasis to the liver his condition became suddenly critical and in the last few days of his life narcotics were again used for general discomfort.

Comment: The extremely gratifying result in this case is felt to result from the limited incision in the frontal lobes. Relief of pain was successful and there were absolutely no changes in intelligence that could be detected. His personality was affected slightly but this was detected only by his physician brother and not by other members of his family.

After the experience with the six cases reported above, particularly Case 6, the author developed a new technic by which a small lesion is made in the lower medial quadrant of the frontal lobe anterior to the ventricle of each hemisphere. This lesion is produced by electrocoagulation.* It was hoped that some of the undesirable results of the conventional procedures might be eliminated by making a small bilateral lesion. Since March, 1949, this operation has been used in 12 cases for the relief of pain. Three of the cases had intractable pain due to advanced carcinoma. They had survival periods of four weeks, six weeks and two months and each patient had relief of pain during the time he lived. Narcotics were no longer necessary and only one of the patients showed any undesirable changes in personality after the first postoperative week. Another case with severe tabetic pain has had marked relief for six months with a unilateral operation. Two cases were complete failures and in each instance the failure is felt to be due to the acceptance of a patient who, in addition to organic pain, had a profound psychoneurosis which was more important than the pain and for which prefrontal lobotomy was not indicated. The remaining six cases

*A detailed report of the operative procedure is being published elsewhere.

with follow-up periods of from one to six months have been successful to date for the relief of pain and none have shown any undesirable side effects.

Discussion

It seems quite certain from the study of these cases that one can offer very little assurance to a patient or his family that unilateral prefrontal lobotomy will produce relief of pain. Recently Scarff[16] reported that in 33 cases of unilateral frontal lobotomy only 66 per cent had successful relief of pain and another 18 per cent had partial relief. Contrary to general opinion, changes of personality and intellect occur after the unilateral operation although they are less severe than after the bilateral procedures. Cases 1 and 3 followed for 15 and 18 months respectively after unilateral frontal lobotomy show distinct changes, though minor, in personality and memory. Therefore, if these operations are to be used, a logical routine would be to perform a unilateral operation originally with the understanding that a similar operation on the opposite hemisphere might later become necessary.

The bilateral isolation of the frontal areas by a standard (radical) lobotomy is very effective for relieving pain and the suffering which goes with pain. In many cases there are marked changes of personality or intellect. In spite of these changes, the procedure is very useful in some cases of incurable illness, particularly incurable carcinoma. This is especially true if morphine addiction is also a problem, since the drug is no longer necessary after surgery and there are no withdrawal symptoms.

Before a bilateral procedure can be offered to patients with pain from non-neoplastic disorders, however, a less crippling procedure must be found. This means that some of the frontal lobe function, which is destroyed in the conventional operation, must be preserved. The new procedure which the author has used offers additional hope toward this possibility. It suggests that the good results of lobotomy, at least those performed for the relief of pain, are not based on a quantitative isolation of the frontal lobes from the remainder of the brain but rather that specific fiber tracts or cortical areas may be destroyed with equal success.

Summary

1. Frontal lobotomy is an effective procedure for the relief of intractable pain..

2. Unilateral frontal lobotomy for the relief of pain is less successful than the bilateral (standard) operation.

3. Unwanted changes in personality and intellect are apt to be severe after the bilateral procedure. These changes, though of less degree, also occur with the unilateral operation performed by standard technic.

4. To broaden the use of this type of operation for intractable pain of noncancerous origin a less disabling procedure is necessary. This may possibly be accomplished by limiting the size of the bilateral operation or by selective bilateral destruction of certain areas of the frontal lobes.

5. A new procedure in which a small symmetrical destructive lesion is produced in the lower medial quadrant of each hemisphere has been successful in relieving pain.

REFERENCES

1. Board of Control, England and Wales, Prefrontal Lobotomy in 1000 cases. London: H. M. Stationery Office, 1947.
2. Freeman, W., and Watts, J. W.: Prefrontal Lobotomy in Agitated Depression. Report of a Case, M. Ann. District of Columbia *5:*326, 1936.
3. Freeman, W., and Watts, J. W.: Pain of Organic Disease Relieved by Prefrontal Lobotomy, Lancet *1:*953-955 (June 29) 1946.
4. Freeman, W., and Watts, J. W.: Prefrontal Lobotomy in the Treatment of Mental Disorders, South. M. J. *30:*23, 1937.
5. Freeman, W., and Watts, J. W.: Retrograde Degeneration of the Thalamus Following Prefrontal Lobotomy, J. Comp. Neurol. *86:*65-93 (Feb.) 1947.
6. Freeman, W., and Watts, J. W.: The Thalamic Projection to the Frontal Lobe. Research Publ. A. Nerv. & Ment. Dis. *27:*200, 1948.
7. Fulton, J. F.: Functional Localization in the Frontal Lobes and Cerebellum, with Particular Reference to the Operation of Frontal Lobotomy. New York: Oxford University Press, 1949.
8. Lyerly, J. G.: Prefrontal Lobotomy and Involutional Melancholia. J. Florida M. A. *25:*225, 1938.
9. Meyer, A.; Beck, E., and McLardy, T.: Prefrontal Leucotomy: A Neuro-Anatomical Report, Brain *70:*18-49 (March) 1947.
10. Moniz, E.: Tentatives Opératoires dans le traitement de certaines psychoses. Paris: Masson et cie, 1936.
11. Penfield, W.: Symposium on Gyrectomy. Part I—Bilateral Frontal Gyrectomy and Postoperative Intelligence, Research Publ. A. Nerv. & Ment. Dis. *27:*519, 1948.
12. Pool, J. L.: Topectomy: A Surgical Procedure for the Treatment of Mental Illness, J. Nerv. & Ment. Dis. *110:*164, 1949.
13. Poppen, J. L.: Prefrontal Lobotomy for Intractable Pain: Case Report, Lahey Clin. Bull. *4:*205-207 (Jan.) 1946.
14. Rinkel, M., et al: The Effect of Bilateral Frontal Lobotomy upon the Autonomic Nervous System, Am. J. Psychiat. *104:*81, 1947.
15. Scarff, J. E.: Unilateral Prefrontal Lobotomy with Relief of Ipsilateral, Contralateral and Bilateral Pain; Preliminary Report, J. Neurosurg. *5:*288-293 (May) 1948.
16. Scarff, J. E.: Unilateral Prefrontal Lobotomy for the Relief of Intractable Pain and Termination of Narcotic Addiction, Surg., Gynec. & Obst. *89:*385, 1949.
17. Scoville, W. B.: Selective Cortical Undercutting as a Means of Modifying and Studying Frontal Lobe Function in Man, J. Neurosurg. *6:*65, 1949.

TREATMENT OF LUNG ABSCESS*

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LUNG abscess is no longer a chronic debilitating disease. The patient with lung abscess frequently can be completely cured in from two to four weeks. No longer is it necessary to have patients with chronic draining sinuses or productive cough following the drainage of a lung abscess. The mortality rate has dropped from as high as 50 per cent in some series reported a few years ago to almost negligible figures in comparison.

DIAGNOSIS

The diagnosis of lung abscess is usually easy. In the acute stage the patient has a high fever, appears sick and is coughing up foul sputum. The sputum, if not putrid at first, usually quickly becomes so. Physical examination of the chest is usually normal even in the presence of fairly large areas of pulmonary involvement. Roentgenographic examination of the chest is necessary to locate the disease. Proper interpretation of the roentgenogram is at times difficult in the acute stage. A cavity with fluid level makes the diagnosis simple, but frequently the cavity has not drained sufficiently to show a cavity containing air, or the cavity is surrounded by pneumonitis to such an extent that it does not show through the area of density.

In the chronic stages of lung abscess the sputum is purulent but frequently has lost its characteristic foul odor. The patient does not appear as ill as in the acute stage. The roentgenogram frequently is more suggestive of atelectasis than of cavity formation.

There are many variations of both the acute and chronic phases of the disease. The sputum is not always foul in the acute stage and may be very putrid after several months of illness. Roentgenograms vary from segmental areas of density to lobar atelectasis in appearance and from single cavities of less than a centimeter to cavities occupying almost an entire lobe or multiple cavities throughout a single lung. It is rare to see patients on a thoracic surgical service with multiple cavities in both lungs.

ETIOLOGY

In the present series of lung abscesses being reported the cause of the infection could be ascertained in very few instances. The

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most usual history was that the patient started running a fever, there was pain in the chest followed by cough and expectoration which was either putrid from the first or rapidly became putrid. The aspiration of vomitus by the alcoholics was thought to be the cause in this group.

The history of patients with lung abscess is different today from a few years ago. In the past it was rare to see a patient with a lung abscess without some definite cause preceding the infection, such as an injury, operation, or pneumonia. It is very possible that the lung abscesses we are seeing today are complications of pneumonia so mild in character as to be unrecognized. Lung abscesses following operations about the face and throat (tonsils) are indeed rare in contrast to the number following such operations a few years ago. It is interesting that this change in the etiology of lung abscesses has taken place coincident with the introduction of chemotherapy and antibiotic therapy.

Treatment

There are three advances in therapy of lung abscesses responsible for the change in prognosis. These are:

First: The free use of transfusions to return the blood volume to normal. There are few infections with which the patient survives that cause as rapid loss of blood, and without external bleeding, as does the lung abscess. Two to three thousand cubic centimeters of whole blood are frequently required to bring the blood volume back to normal. Before the blood volume changes in infection were well understood the patients were transfused until the erythrocyte count was five million. The percentage of cures obtained in small acute lung abscess with conservative treatment alone was about the same before chemotherapy and antibiotic therapy as it is today. We now transfuse until the hematocrit is 50 per cent. This is based on the work of C. Lyons on chronic infections in which he pointed out that the blood volume was usually about normal if the hematocrit was brought up to 50 per cent. Hematocrits are easily obtained in contrast to the determination of the blood volume, and clinically this technic has been satisfactory.

Second: The use of antibiotics has facilitated greatly the preoperative preparation of the patient although it is doubtful if the morbidity or cure rate of the small acute lung abscess has been altered greatly by these agents. The sulfanilamides seemed to have little if any effect on lung abscesses. Penicillin in doses of 100,000 units every three hours intramuscularly has been very effective in reducing the amount of sputum as well as quickly ridding the sputum

of its offensive odor. Penicillin in the forms given once or twice daily has not been effective. Streptomycin combined with penicillin has been of doubtful additional value. Aureomycin promises to be an effective agent but has been used in too few cases to evaluate at present.

Third: The change in surgical technic from drainage of the lung abscess to excision of the abscess has done much to increase the cure rate.

Report of Cases

Since January, 1946, 61 patients with lung abscess have been treated by pulmonary resection. Three patients died, a mortality rate of 5 per cent. Fourteen patients had pneumonectomies. Three of the pneumonectomies developed empyemas and required subsequent thoracoplasties to obliterate the empyema space. Only one of the pneumonectomies had a recognizable bronchopleural fistula. There were only three segmental resections in the group. Several segmental resections were done along with lobectomies where the infection had extended across the interlobar fissure. Of the 58 patients who survived the operation all are well except one who died several months postoperatively of a diabetic condition.

The ages of the patients varied tremendously, the youngest being three years of age and the oldest 73. Sixteen patients were 50 or over. Five of the patients were 10 years of age or less.

Concerning the three deaths, one occurred three hours after a pneumonectomy due to a tie slipping off the pulmonary artery. One death occurred in a 15 year old boy due to bleeding from the severed adhesions on the chest wall. The bleeding started after the operation had been completed, probably due to a mismatching of blood. Another boy of 10 developed pulmonary edema after being moved back to the room. Of the 23 patients done this year there has not been a death.

Discussion

All patients admitted with lung abscess are treated with antibiotics, usually 100,000 units of penicillin every three hours intramuscularly. Daily transfusions of whole blood (500 c.c.) are given until the hematocrit reaches 50 per cent. The patient is kept in bed but postural drainage, thought so important in the past, has not been used. A lung abscess either drains or does not drain into the bronchial tree depending on the degree of patency of the bronchus leading to the abscess. The position of the patient has little if anything to do with this drainage. Pus reaching the normal bronchial tree is promptly coughed up.

The use of this regimen usually produces rapid clinical improvement. The temperature rapidly returns to normal. The sputum decreases markedly in amount and loses its putrid character. Roentgenograms of the chest are taken at weekly intervals.

Single cavities of less than 3 centimeters frequently have been healed by this method in two to three weeks. If the pneumonitis and cavity have not improved markedly by the end of two weeks the abscess usually will not heal under prolonged conservative treatment and should be resected. Usually the cavity of less than 3 centimeters will almost have disappeared at the end of one week. At the end of the second week only a small area of density remains which rapidly disappears. The failure of three such small cavities to heal accounted for the three cases in which segmental resections were done in this series.

A review of our medically treated cures was not included in this paper because it is believed most lung abscesses either heal spontaneously or heal under the treatment of the family physician. The abscesses coming to the thoracic surgical service mostly were failures to obtain cure by the family physician.

Single cavities of over 3 centimeters in diameter rarely healed. At times it was possible to sterilize the cavity but even then healing did not take place. Such cavities always become re-infected although the re-infection may not take place for several years. One cavity of 10 years' duration was resected due to massive hemorrhages, although there were no symptoms of infection. The hemorrhage was apparently caused by the cavity being distended by a pure culture of aspergillus. Another similar patient was recently operated on with similar findings, but the duration of the cavity was not known in this case.

The larger cavities have been treated by lobectomy because of the amount of lung involvement. Examination of these lobes has shown that a segmental resection of the cavity would have left considerable bronchiectasis in the adjacent portions of the lobe. For this reason our policy has been to reserve segmental resection for the acute small abscess that fails to heal on the conservative treatment within two weeks' time. Our policy now is to do a lobectomy for the cavity of over 3 centimeters in diameter as soon as the patient can be put in condition for surgery. The importance of bringing the blood volume to normal and the subsidence of toxicity for a few days preoperatively cannot be overemphasized. The properly prepared patient withstands pulmonary resection without any difficulty.

Patients with multiple abscesses throughout one lobe or involving

an entire lung cannot be treated successfully medically or by any other means short of resection of the involved lung.

Two patients with lung abscess have been drained during this period. The reasons for draining these patients were that they were elderly patients with congestive heart failure in which pulmonary resection was definitely contraindicated. Both patients are dead.

Bronchopleural fistulas have not been a problem in this series of resections. Empyemas following lobectomy have occurred so rarely and were so small that this complication was of negligible importance. The three empyemas following pneumonectomy were the most serious complications, requiring complete thoracoplasties. The lobectomies rarely required hospitalization as long as two weeks following the resection. The patients were able to return to their former occupations within a matter of days following their discharge from the hospital, free of cough, expectoration, and draining sinuses.

Conclusions

1. Sixty-one patients with lung abscess treated by pulmonary resection have been reported.

2. The mortality rate of the operation was 4.9 per cent.

3. Patients treated by resection make complete recoveries from the infection and are able to return to work quickly.

4. The bronchiectasis associated with lung abscess is removed along with the abscess.

ACQUIRED MEGACOLON

Report of a Case in a Seventy-four Year Old Male

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MEGACOLON can be divided into two types: (1) that due to a demonstrable obstruction and (2) that in which no obstruction can be demonstrated. Those which are clearly obstructive may occur in either children or adults. Those which are clearly nonobstructive are found most frequently in children and when present in adults, the history of the disease goes back to childhood.

The nonobstructive cases are usually referred to as "Hirschprung's disease." Hirschprung, as quoted by Finney,[1] termed these cases "Congenital Idio-pathic Dilatation of the Colon" and contended that they were not due to obstruction and that they occurred only in children. Megacolon occurring in adults, he called "Pseudomegacolon."

There is another group of cases in which a "functional obstruction" is said to be present. The obstructions are supposed to be due to a redundant sigmoid and mesosigmoid which allows kinking, or due to mucosal valves. This group has caused considerable confusion in nomenclature in the literature. Some of these have been reported as cases of Hirschprung's disease occurring in adults. Other writers contend that because of the obstructive element they are not true cases of Hirschprung's disease. Since the report of Wade's case by Royle[2] in 1927, in which a cure was effected by lumbar sympathectomy, there is a tendence to limit the term to cases in which there is a disarrangement of the automatic nervous system. In adult cases reported in detail, one is struck, as was Finney,[1] by the high incidence of obstructive factors.

The following case presents a definite but unusual type of obstruction of the sigmoid colon which led to a dilatation and hypertrophy of the colon or "pseudomegacolon."

D. McC., a 74 year old white male farmer, was admitted to the Louisville General Hospital Jan. 1, 1947, because of abdominal pain and distention, vomiting, and no bowel movements.

The onset followed a large Christmas dinner six days before admission. Pain and vomiting subsided twenty-four hours after the onset. His abdomen then became progressively more distended. He had not passed gas or had a bowel movement from time of onset until admission to the hospital. After the

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third day of his illness, he could no longer reduce his left inguinal hernia. The hernia was not painful or tender.

Nineteen years before admission, while using a cross-cut saw, he had been seized with a severe pain in his left groin which caused him to roll around on the ground. He noticed for the first time a mass in his groin. After two days of pain he was able to reduce the mass and return to work. He had no vomiting during this episode. Until about five years previously the hernia would come down most of the time but could always be easily reduced until the present illness. The patient said that he had never worn a truss.

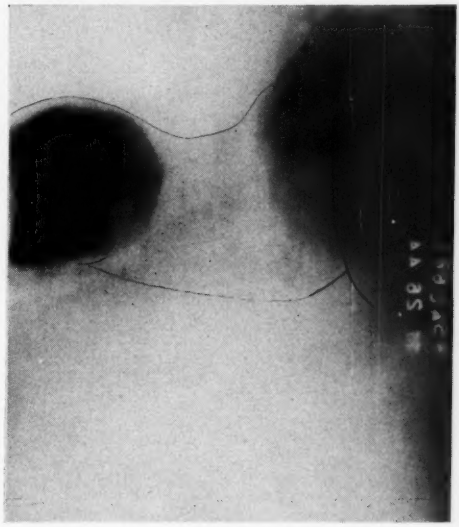

Fig. 1. Flat film of abdomen in November, 1944 (2½ years before present admission). Marked distention of colon.

He stated that his bowels had usually moved once a day all of his life and that he took a cathartic only once or twice a year for mild constipation. However, there had been two previous admissions for dermatitis, one in 1944 and again in 1945, at which times abdominal distention was noted on the physical examination and in 1944 gaseous distention of the colon was demonstrated by roentgenogram (fig. 1). Thus, we know this distention had been present at least three years before the present admission in 1947.

At the time of both previous admissions there was edema, mild cyanosis and exfoliating skin lesions of both legs due to venous stasis. No varicosities were present. The stasis was probably secondary to abdominal distention.

At the time of the admission in 1947 the patient was a well developed, well nourished white male, not appearing acutely ill. Temperature was 100°, and blood pressure 120/70. The abdomen was distended, tympanitic and moderately tender. Few peristaltic sounds were heard. No fluid waves or shifting dulness were found. Rectal examination showed external hemorrhoids. There was a moderately large left inguinal hernia which was not tender and was reducible. The red blood cell count was 5,010,000. There were 13 Gm. of

hemoglobin per 100 c.c. The white blood cell count was 9,650 with a normal differential count. The urine analysis was negative. Roentgenogram of the abdomen showed marked gaseous distention of the colon throughout its proximal three fourths (fig. 2).

Fig. 2. Flat film of abdomen taken on day of present admission (January, 1947). Marked distention of colon.

Intestinal suction by a Miller-Abbott tube and hot rectal irrigation and enemas were not effective in reducing the distention. A barium enema was done and there was noted a constant narrowing in the sigmoid. This suddenly relaxed with low pressure and much barium passed into the remainder of the colon and considerable gas was passed per rectum at the same time. The sigmoid colon at the time of the fluoroscopic was in the pelvis. Films made immediately (fig. 3) following the enema and postevacuation (fig. 4) showed marked dilatation behind a narrowing at the sigmoid colon. The colon was no longer in the pelvis but was directed inferiorly across the top of the pubis in the region of the left inguinal hernia. No evidence of carcinoma was seen. The narrowing of the colon was thought to be due to constriction of the herniated loop of sigmoid colon in the hernial sac.

Because of continued distention and inability of the patient to pass gas or feces by rectum, the abdomen was opened through a McBurney incision forty-eight hours after admission. The cecum was greatly dilated but not under tension. Because of the lack of tension it was thought safe to explore the pelvis. The wall of the cecum was about twice normal thickness. The descending colon was widely dilated but under no tension and had a thickened wall.

The dilation was traced down into the hernia. The bowel was pulled from

Fig. 3. Barium enema showing colon entering hernial sac.

Fig. 4. Post evacuation film showing marked dilatation of colon.

the inguinal canal and no definite point of obstruction or tumor mass could be found. There was a gradual decrease in diameter to the size of normal empty

sigmoid past the segment which had been contained in the hernia. Several loops of the sigmoid were adherent to each other.

A cecostomy was done at this time. Following this there was little general change in the patient's condition. Repeat roentgen examination of the abdomen showed little change in appearance the next day.

On Jan. 9, 1947, a spinal anesthetic was given without the use of vasopressor drugs. Analgesia was present as high as the third thoracic dermatome. Films were made at 20, 35, 65 and 95 minutes following administration of the anesthetic. No evacuation or marked contraction of the colon was noted.

On January 14, the abdomen was explored through a left rectus incision. The sigmoid colon was found in the left inguinal hernial sac from which it was pulled without difficulty. This loop was bound together with thick adhesions which had produced a segment of kinks and partial obstruction for a distance of 10 cm. The colon proximal to the constricted area was much thicker than normal and was markedly dilated, but under no tension. Distal to the area of obstruction the colon was of normal size and thickness.

Fig. 5. Barium enema taken 4 months after operation. Colon is normal.

Enough of the adhesions were broken up to insure an adequate sigmoid lumen. The hernia sac was amputated, ligated and repaired from within. On the third day, the patient began to pass gas by rectum and two days later had a normal bowel movement. The cecostomy closed spontaneously by the ninth day and the patient was discharged on the tenth day in good condition and with no complaints.

At two and four months after operation, he was having daily bowel move-

ments, but was slightly distended. Barium enema (fig. 5) showed some decrease in the size of the colon. In June, 1949, two and one-half years after operation, he was completely symptom-free and a barium enema showed that the colon had returned to normal (fig. 6).

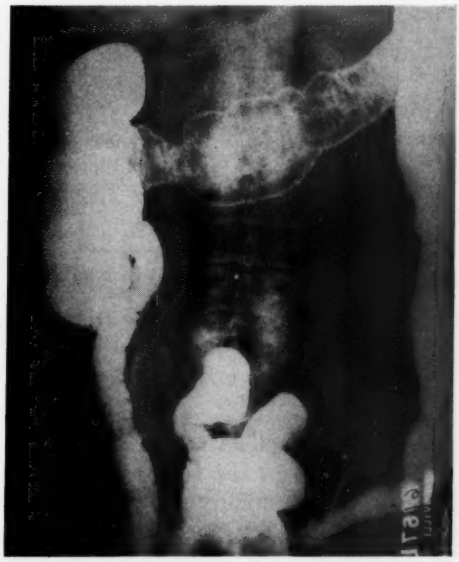

Fig. 6. Barium enema 2½ years after operation. Colon remains normal.

Whether or not true megacolon occurs in adults (i.e. without any obstructive element) it is clearly shown by this case that a long-standing partial obstruction can produce dilatation and hypertrophy of the colon, i.e. pseudomegacolon.

This case might be classified as acquired, secondary or as pseudomegacolon (Finney). The obstruction was due to old constricting adhesions binding the sigmoid to itself and not by a constriction of any part of the hernial sac. It was adherent posteriorly only to a small area of the sac. The hernial sac contents could be reduced at any time before operation but apparently were reduced as a self-adherent mass. At operation the adhesions did not prevent all the contents from being easily pulled up into the abdominal cavity.

Hypertrophy and chronic dilatation of the colon due to obstruction is rarely observed. This is probably due to the rareness of lesions which will produce a high grade incomplete obstruction in the colon for years. Carcinomatous obstruction will produce an acute dilatation but not hypertrophy because of the time limit involved by the progressive nature of the lesion. However, Rankin[3]

reported three cases of megacolon resembling the acute dilatation produced by the obstruction of carcinoma of the sigmoid. The obstruction due to diverticulitis is usually relieved in a relatively short time. Wangensteen[4] states that adhesive obstruction rarely involves the colon and that when it does, it is due to compression as by a band running over the transverse colon to a fixed point. This usually causes an acute episode of complete obstruction.

It is unusual for the colon to form the major part of the content of an inguinal hernia. Watson[5] could collect only 800 cases in the literature until 1924. In these 800 cases the order of frequency was cecum, sigmoid, ascending, descending and transverse colon. Frankau[6] found in 654 strangulated inguinal hernias that large bowel was present in 21. Six were of large bowel alone. Seven were made up of large bowel and omentum and eight were sliding hernias.

Megacolon occurs most frequently in children but apparently can occur at any age. Our patient is older than any we can find in the literature. Johnson[7] reported a male, aged 60; Rivers[8] a male of 53 and Eiss[9] a male, aged 59.

The part of the colon involved varies in different cases. Grimson[10] divided the variations of the sites of involvement into three groups. Group I—Uniform involvement of all of the colon and a dilated or easily dilatable rectum. Group II—Dilation of the proximal colon terminating in the sigmoid region in a normal segment of sigmoid. Group III—Enormous dilatation of the descending and upper sigmoid colon.

Our case falls into Group II. This group includes about one fifth of the cases: 32 of 169 cases of megacolon reported by Neugeberger,[11] 4 of 26 reported by Whitehouse,[12] and 7 of 24 reported by Grimson[10] fall into this group. Most of these cases were in children.

Conclusion

1. A case of hypertrophy and dilatation of the colon, from cecum to the midsigmoid colon in a 74 year old white male is reported.

2. The enlargement of the colon was due to adhesive obstruction of the sigmoid colon. The adhesions were caused by a large longstanding left indirect inguinal hernia.

3. Incomplete obstruction existing for a period of years can cause second or pseudomegacolon.

BIBLIOGRAPHY

1. Finny, J. M. T.: Congenital Idiopathic Dilatation of the Colon, Surg., Gynec. & Obst. 6:624-643, 1908.

2. Royle, N. D.: The Treatment of Spastic Paralysis by Sympathetic Ramisection, Proc. Roy. Soc. Med., London *20:*1799-1804, 1927.
3. Rankin, F. W.: Megacolon Secondary to Carcinoma of the Sigmoid, Report of 3 Cases, S. Clin. North America, 863-874 (Aug.) 1929.
4. Wangensteen, O. H.: Intestinal Obstruction, 2nd ed., Springfield: Charles C. Thomas, Publisher, 1942.
5. Watson, L. F.: Hernia. St. Louis: C. V. Mosby Co., 1924, p. 500.
6. Frankau, C.: Strangulated Hernia, Review of 1,487 Cases, Brit. J. Surg. *19:*176-191, 1931.
7. Johnson, P. P.: Megacolon in the Adult with Report of Two Cases, Boston M. & S. J. *184:*88-93, 1921.
8. River, L. P., and Guber, J. A.: Transverse Megacolon Associated with Chronic Volvulus, Ann. Surg. *117:*786-790, 1943.
9. Eiss, S.: Megacolon (Hirschsprung's Disease): Treatment by One-Stage Extraperitoneal Resection, Am. J. Surg. *34:*272-285, 1936.
10. Grimson, K. S.; Vandegrift, H. N., and Dratz, H. M.: Surgery in Obstinate Megacolon, Surg., Gynec. & Obst. *80:*164-173, 1945.
11. Neugebauer, F.: Die Hirschsprungsche Krankheit. Ergebn. d. Chir. u. Orthop. *130:*598-670, 1932. (Ibid) *7:*598-670, 1930.
12. Whitehouse, F.; Bargen, J. A., and Dixon, C. F.: Congenital Megacolon, Favorable End Results of Treatment by Resection, Gastroenterology *1:*922-937, 1943.

TUMORS OF THE RECTUM

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In this short discussion of "Tumors of the Rectum," were I to classify them I would simply be rehashing classifications which can be found in the different textbooks. Therefore, I will confine my remarks to the diagnosis of benign and malignant tumors, wishing to stress the importance of rectal examination.

The simple adenoma is the most common type of benign tumor found in the rectum and sigmoid. In the early stages it appears as a slight elevation of the mucosa, later it may assume a lobular shape and may remain sessile or become pendunculated. While it may not produce any symptoms, bleeding is perhaps the most common. Other symptoms may be constipation, straining at stool if the tumor is in the lower rectum, and protrusion of the tumor from the anal canal if there is a long pedicle. It is estimated that about 25 per cent of solitary adenomas become malignant.

I think it is now universally agreed that the multiple adenomas or polyposis are inherited as a Mendelian dominant and in Rankin's[1] opinion "malignant changes invariably result if the individual lives long enough."

There is the second variety of multiple adenomas which by some is described as pseudopolyposis and which follows inflammatory disease, especially chronic ulcerative colitis. These pseudopolyps may also undergo malignant degeneration. It has been said that about 12 per cent of all malignant tumors originate in the anus, rectum and sigmoid and that approximately 75 per cent of all intestinal malignancies occur in this area and therefore can be detected by digital and proctosigmoidoscopic examination. Malignancy of the anus, rectum and sigmoid is next in frequency to that of the stomach and equal to that of the uterus.

Too often we hear the remark: "I had a G. I. series on this patient and it is negative," thus giving the physician a feeling of false security. I know that the roentgenologists agree with me that a rectal examination including a proctosigmoidoscopic examination should precede the x-ray examination whenever there is a history of bleeding from the rectum or if there is any other indication for a rectal examination. Estimates by roentgenologists as to the number of rectal tumors, or the percentage of rectal tumors, diagnosed by x-ray examination vary—some feeling that they can detect from 25 to 50 per cent; however, since there is no follow-up in many of these cases I think it is very difficult to determine how many are

missed. I am sure we all agree that a small tumor in the ampulla of the rectum is more often missed than found by x-ray examination. Bacon[2] states that a barium enema study is of value in diagnosing tumors above the rectosigmoid, but of little use in the rectum proper.

The following brief case report illustrates the importance of a rectal examination which should have been made before the roentgenologist's examination. The patient, a white male, aged 59, stated that for the past seven months he had had attacks of diarrhea, alternating with constipation, anorexia, weakness, and loss of weight (from 200 to 125 pounds). During the preceding few weeks he had 15 to 18 dejections a day which consisted mostly of mucus and at times a small amount of bright red blood. He attributed his condition to nervous exhaustion. His past history was negative except that he had had several nervous attacks, the result of overwork as an executive. The family history was negative for tuberculosis and cancer. The patient had just undergone a complete physical examination including a complete gastrointestinal x-ray. Upon rectal examination a large growth was found, the lower limits of which were only an inch above the anal sphincter. The diagnosis: Cancer of the rectum. Had a rectal examination been done the diagnosis would have been made earlier.

Conclusion

1. Whenever there is an indication for a rectal examination a proctosigmoidoscopic observation should precede the roentgenologic examination.

2. A barium enema (including double contrast study) is a "must" in diagnosing tumors above the rectosigmoid, but is of little value in the diagnosis of tumors in the rectum proper.

BIBLIOGRAPHY

1. Rankin, F. W.: Surgical Treatment of Polyps of the Colon, Postgrad. Med. 5.
2. Bacon, H. E.: Essentials of Proctology, Philadelphia: J. B. Lippincott Company, 1943, page 199.

PATHOLOGIC PICTURES OF ANESTHETIC COMPLICATIONS*

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No type of anesthetic procedure is free of complications. Some of these conditions are incompatible with life, but most are only temporarily embarrassing. Deaths are relatively few, and the seeming hesitancy of operating room teams of the past to come face to face in the autopsy room with the consequences of their unavoidable catastrophes or possible lapses has sharply limited the amount of available accurate pathologic data. In this paper, we shall describe briefly the more common complications and their pathologic pictures.

OXYGEN WANT

A deficiency of oxygen is the most frequently encountered and potentially the most serious complication of general anesthesia. Indeed, it is sometimes a *part* of the anesthetic procedure, rather than a complication, since many anesthetists make use of the soporific powers of lowered oxygen tensions. All of the organs of the body suffer, but the most important pathologic changes are found in the brain. These are due to both nerve cell injury and vascular damage. They are expressed in generalized fashion through postoperative psychoses, feeble mindedness, confusional and comatose states, and personality disorders. Focal manifestations include hemiplegia, blindness, aphasia, convulsions, choreathetosis and decerebrate rigidity. Cyanosis is *not* a reliable danger signal, since it begins to lose its value when the hemoglobin concentration falls below 80 per cent. With a hemoglobin value below 60 per cent, *any* degree of cyanosis indicates serious trouble, and a person with a value below 40 per cent may die of anoxia without *ever* showing cyanosis. The neuron reactions to oxygen deficiency vary with the metabolic rates of the cells; those cells of the most recent phylogenetic origin having the highest metabolic rates and showing the most damage. The pyramidal cells of the cerebral cortex are said to show irreversible changes after eight minutes of complete anoxia, while the Purkinje cells of the cerebellum may survive for thirteen minutes. Cells in the medulla may continue to function after twenty to thirty minutes of complete anoxia. Microscopically, one sees all degrees of change from rapid lysis to slow dechromatization. Patchy necro-

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sis of the cerebral pyramidal cells is sometimes shown after only a few minutes, if the sections are carefully studied.

The circulatory changes consist of stasis, edema, diapedesis of red cells, hemorrhage, infarction and softening. Circulatory damage is greatest in the basal ganglia and internal capsule. It is also severe at the junction of the gray and white matter of the cortex, where the arteries take an angle turn.

Once the process has begun, the metabolism of the cells seems to be actually increased by the oxygen deficiency, as evinced by the convulsions which sometimes occur. The resulting local acidosis, combined with the stagnant anoxia, further increases the local dilatation and oxygen want. The cycle is repeated more and more rapidly with each passing moment. Should the patient die suddenly on the operating room table, nothing may be seen at autopsy, since it takes from hours to days for the histologic picture to develop. These patients show, usually, only minute petechial hemorrhages in the brain, meninges, and various organs. In patients who have partially recovered and lived for some time, proliferation of the glial components, areas of cystic softening, and meningeal adhesions have been found.

Cardiovascular Lesions

Cardiovascular lesions encountered during anesthesia are usually the result of hypertension, hypotension, direct action of the agent on the tissues, or embolic phenomena.

Hypertensive accidents are for the most part hemorrhagic in nature. A hypertensive nosebleed during the excitement stage may interfere with the conduction of an inhalation anesthetic, while a cerebral hemorrhage or rupture of an aneurysm of a large vessel may abruptly terminate any type of anesthetic procedure. These accidents are more common in middle aged or elderly patients suffering from atheromatous arterial disease. The commonest sites of the cerebral hemorrhages are the internal capsule and lenticular nucleus, since the small arteries to these structures come directly from the middle cerebral arteries, with no "stepdown" of pressure. Next in frequency are the vessels of the pons and medulla. The affected brain tissue is disrupted and destroyed. A clot is found at the site of the hemorrhage, and this may be surrounded by petechial hemorrhages.

Rupture of an aneurysm is also to be feared during a stormy induction, a violent reaction to endotracheal intubation, or during the period of hypertension which may follow the successful injection of a vasopressor agent and the unsuccessful injection of a spinal anes-

thetic agent. Syphilitic or arteriosclerotic aneurysms of the great vessels rarely rupture at this time, but intracranial aneurysms may do so. In young people, these are usually of the congenital type, at the base of the brain. In older patients, the miliary type of aneurysm is encountered. These are false aneurysms, formed by small hemorrhages into the adventitia of the vessel through atheromatous plaques.

Acute coronary insufficiency may follow periods of hypertension, with no definite evidence if death is sudden, and areas of myocardial infarction if death is delayed. The anterior descending branch of the left coronary artery is the most frequent sufferer. The same picture may be seen with hypotension, usually when local or regional anesthetic agents are used (since oxygen-rich mixtures are often given when general anesthetic agents are used).

Hypotensive accidents are encountered more frequently during local, regional and intravenous anesthetic procedures than during inhalation anesthesia, although those sequelae from prolonged shock may occur after any type of procedure. The organs most severely affected are the brain, heart, and kidney. In obstetric anesthesia, hypotension is to be avoided, since the uterine tone may be sufficiently strong to overcome the arterial pressure and the fetus may die from oxygen want.

Cerebral or coronary thromboses may be unfortunate sequelae to prolonged hypotension. The clot may propagate after the condition has been corrected, leading to infarction of much needed brain or heart tissue. Renal function is, as a general rule, seriously impaired should the arterial pressure remain under 70 millimeters of mercury for long periods of time. After several hours of hypotension, the cloudy swelling of the mild cases may be replaced by renal cortical necrosis much resembling that seen after poisoning with bichloride of mercury or a hemolytic transfusion reaction.

Toxic cardiovascular accidents are most commonly encountered with the halogenated agents (chloroform, ethyl chloride, tribromethanol, trichlorethanol, trichlorethylene) and with cyclopropane. The cardiac manifestations, enhanced by excess endogenous or exogenous epinephrine, are disturbances in rhythm, the pacemaker being displaced progressively downwards to ventricular fibrillation. Should the heart be observed during a period of ventricular fibrillation, it will be seen to be soft, flabby, somewhat dilated, and twitching feebly. One must always remember, however, when blaming the anesthetic agent for this catastrophe, that one of Simpson's first patients died suddenly while breathing through a gauze mask *preparatory* to a chloroform induction. Hertzler also claims to have

seen a *bystander* drop dead as the first injection of a local anesthetic agent was made.

A direct toxic effect is exerted upon the myocardium by the halogenated agents when any concentration sufficient for anesthesia is present, but the autopsy findings after operating room deaths consist only of cloudy swelling of the fibers and small pericardial hemorrhages.

Intravenous anesthesia has been the cause of painful chemical phlebitis, the strongly alkaline barbiturate solutions not being sufficiently diluted with blood. Embolic phenomena are not common here since the clot is usually adherent. Perivascular infiltration has eventuated in local necrosis and slough. Intra-arterial injection has been followed by severe spasm of the vessels and gangrene of the part (this complication may possibly be prevented by keeping the needle in place and immediately injecting a few cubic centimeters of one per cent procaine solution).

Embolic phenomena are usually due to fat, gas, and clotted blood. Gas embolism is often a complication of the surgical procedure, a vein being opened in such a manner that collapse does not occur, and air is sucked into the vein. Straining respirations, partial obstruction, and the use of cuffed endotracheal tubes with too small a bore can greatly increase the amount of air drawn into the vein (by increasing the negative intrathoracic pressure). Forcing blood transfusions with air pressure or oxygen under pressure has also caused several deaths from gas embolism. The patient may die an immediate anoxic death, recover completely, or recover with visual or central nervous system sequelae. Air may sometimes be seen in the retinal vessels by ophthalmoscopic examination. The skin is irregularly blanched due to capillary obstruction by the bubbles. The heart is usually full of bloody froth. The most dramatic picture is seen when pressure transfusions are continued after the bottle is empty of blood, and great quantities of froth come foaming into the operative field through the vessels in the operative site. Causes of death include acute cerebral anaemia, multiple cerebral gas emboli, and coronary gas emboli.

Fat embolism is occasionally encountered during surgery on the long bones, especially in fractures of the femur. A case has also been reported following the administration of intravenous ether after a heavy meal. The patients are literally smothered to death, the fat particles plugging the pulmonary capillaries. Examination of the brain may show multiple petechiae with a fat particle at the center of each tiny hemorrhage. All of the organs of the body contain the globules of fat in their tissues, and urinalysis or sputum examination may be diagnostic.

Blood clots loosed during surgery do not pass through the pulmonary circulation, but remain in the vessels of the lung (paradoxic embolism has not yet been reported as a complication). Depending upon the size of the vessel occluded, the patient becomes cyanotic to varying degrees, and may present unexplained shock. Pulmonary emboli should be borne in mind when patients who are good candidates for the phenomenon become suddenly cyanotic and remain so in spite of apparently intact circulation and easy inflation of both lungs with mixtures known to contain adequate oxygen.

Pulmonary Lesions

Pulmonary lesions are traditionally classed as inhalation anesthetic complications, although age, position on the operating table, the skill of the anesthetist and the type of operation are far more important in their development than is the type of anesthetic procedure. The more serious complications are bronchopneumonia, aspiration pneumonia, atelectasis, tuberculous spread, lung abscess, pneumothorax, and pulmonary edema.

Bronchopneumonia is also called lobular pneumonia, because of its patchy character. Scattered bronchioles become filled with inflammatory secretions. The trapped air is absorbed and the lobules collapse. The neighboring alveoli are filled with an inflammatory exudate consisting of polymorphonuclear leukocytes, a few cells, and fibrin. The consolidated areas alternate with areas of congestion, collapse and compensatory emphysema. Recovery is often not complete, and bronchiectasis may follow.

Aspiration pneumonia may follow the inhalation of infected material, usually during operations on the mouth, sinuses or tonsils. It is also seen after the inhalation of vomitus or foreign bodies. Atelectasis, suppuration and abscess formation are common. Aspirated vomitus may set up a chemical pneumonitis. The right lower lobe is the usual wastebasket for material dropping into the tracheobranchial tree, but all lobes have been involved. In a clinical examination, the right lower lobe may often seem involved when it is not, due to the position of the liver just below, and the apprehensive state of mind of the examining anesthetist.

Abscesses begin as solid masses of inflammatory tissue. Liquefaction occurs, and a cavity filled with pus eventuates. The aspiration abscesses usually communicate with a bronchus. The abscess wall is ragged and necrotic at first, but in chronic cases becomes thickened and smooth and hard.

Tuberculous spread is a feared complication of anesthesia and of any type of surgical procedure in tuberculous patients, but it is

most common after thoracic procedures. The incidence is about the same with any type of general anesthesia. Some cases have been reported after local and spinal anesthesia. (In evaluating reports, it must also be remembered that spreads occur where there has been no anesthesia and no surgery.) A tubercular pneumonitis first appears, the spread being bronchiogenic in type. The fatal cases show exudation, multiple areas of consolidation, and multiple small abscesses.

Laryngeal or tracheal tuberculosis is a feared complication of endotracheal anesthesia in tubercular patients, especially when a cuffed endotracheal tube is used, but no positive evidence has as yet appeared in the literature.

The term "pulmonary atelectasis" is used to describe collapse of a portion or all of a lung. There are three types—compression, infantile, and obstructive, of which only the last concerns us here. Obstructive atelectasis depends for its formation upon two factors —accumulation of secretions and diminished respiratory excursions. Air in the affected portion is absorbed, that part of the lung collapses, and should the obstruction not be relieved promptly, pneumonitis and abscess formation may follow.

Patients may develop atelectasis on the operating table, especially if they are placed in embarrassing positions and anesthetized with agents which further depress respirations. Cyclopropane, pentothal-oxygen, and curare are the chief offenders. The oxygen rich mixtures accompanying these agents can mask a gradually developing lower respiratory obstruction until the patient is again breathing air (the condition generally becoming apparent about the time the patient is placed in bed). The atelectatic tissue is firm, airless, steel blue or slate gray (depending upon the amount of circulatory congestion) and sinks when placed in water. Microscopically, the alveolar walls are pressed firmly together, and the vessels are congested. In severe cases, the trachea, mediastinum and apex impulse are deviated toward the affected area, and there is rapid onset of tachycardia, dyspnea and cyanosis.

Pneumothorax is a complication of both general and regional anesthesia. In general anesthesia it is most often the result of forceful inflation of the lung during resuscitation from apnea (especially after overdosage of curare), or during routine reinflations involved in thoracic surgery. An emphysematous bleb or a surgical suture line may give way. Surgical procedures around the diaphragm occasionally become intrapleural, sometimes unbeknownst to both anesthetist and surgeon. Brachial block, stellate block, paravertebral, intercostal and splanchnic blocks all carry this hazard. The

air enters the pleural cavity from without or from within (or both). There is much evidence to show that the major part of the air may sometimes enter through the visceral pleura, since it is difficult to penetrate the parietal pleura without also penetrating the visceral pleura and lung. The sudden production of a large pneumothorax may be one cause of "pleural shock." Ordinarily, the amount of air present is not large enough to produce serious symptoms. The air is gradually resorbed and the collapsed pulmonary tissue reexpands. On rare occasions, blood may collect, organize, and make reexpansion impossible. Secondary infection results in empyema.

Tension pneumothorax is a less common condition. Injury to the lung may allow air to be forced into the pleural cavity during expiration. A rent which cannot seal off, or a torn bronchus may add air to the pneumothorax with each respiratory cycle until the entire lung is collapsed and pressure on the mediastinum begins. Should this be a postoperative or post-traumatic complication, the air may be forced through the chest wound into the tissues, where it may dissect for a considerable distance. It is easily palpated over the thorax, abdomen and neck, imparting a spongy, crackly feel to the tissues.

Pulmonary edema is encountered in cardiac patients, shocked patients, in those suffering from chronic respiratory obstruction, in gas and blast casualties, and in those who have survived an anesthetic explosion. In the mechanical type of pulmonary edema, the fluid is clear or somewhat bloody, while in the inflammatory type it is filled with organisms, desquamated and necrotic cells, and fibrin. The lung is voluminous, heavy, doughy or hard, and will pit on pressure. Steady pressure will force out the fluid, leaving the lung soft.

Endotracheal Anesthesia

Endotracheal tubes and laryngoscopes may be harmful as well as helpful. Lips, gums, teeth, palate, pharynx, larynx, trachea, and esophagus all suffer. Deviated septa, turbinates, and nasal polpi are abraded by nasotracheal tubes. A laryngoscope blade prowling about the pyriform sinuses may explore the deeper tissues of the neck. A tightly inflated cuff may produce local ischemia of the tracheal wall, with eventual necrosis and mediastinitis. An endotracheal tube can easily lose its advantages if it is too small, becomes kinked, filled with blood or mucus, has too lengthy or complicated connections with the anesthesia machine, or slips into a main bronchus.

The subglottic area, especially in children, is richly vascular and

may become edematous if sufficiently irritated. In children, the upper part of the respiratory tract develops more rapidly than the lower portion. A tube passing through the nose may not enter the larynx, and tubes passing through the larynx may jam in the trachea just below. Ulceration, perforation, and stenoses have eventuated.

Although puncture wounds, broken teeth, laryngeal granulomata, subglottic and glottic edema, mediastinitis and mediastinal emphysema are the more spectacular sequelae of endotracheal anesthesia, they are not common.

Donnely, Grossman, and Grem have recently shown that 60 of 100 routine intubations by first-year residents could be classed as traumatic. Forty patients showed congestion of the vocal cords or membranous glottis; 20 showed ecchymoses, contusions or edema of the tonsils, palate, and tongue. There were three submucosal puncture wounds of the larynx, and two epiglotti were crushed and edematous. None of the injuries, however, more than temporarily interfered with function.

Gastrointestinal Lesions

The gastrointestinal tract suffers from both anesthesia and surgery. It is not safe from the anesthetist even when he resorts to regional procedures. Esophageal punctures have followed sympathetic and splanchnic blocks, the patients occasionally dying of mediastinitis. Abdominal field block may result in a perforated bowel and distressing postoperative peritonitis. Caudal block has failed because the solution has been deposited in the rectum. The same has been true of transsacral block—the needles penetrating the anterior sacral foramina. Any perforation of the gastrointestinal tract through the thorax, back, or buttocks is likely to result in severe infection along the needle path, with abscesses difficult to approach and treat.

Spinal anesthesia, by permitting maximal contraction of the bowel, may rupture the bowel above a point of obstruction, or cause perforation of a gangrenous appendix. The same contraction may suddenly expel large quantities of intestinal and gastric secretions into the pharynx and trachea when spinal anesthesia is given in cases of intestinal obstruction, embarrassing both the patient and the anesthetist. After a spinal anesthetic is administered, a perforating peptic ulcer may become a perforated peptic ulcer.

Cole, administering curare to dogs (in large doses), noted a bloody diarrhea in several cases. Two dogs which were autopsied presented severe mucosal hemorrhages, with a jelly-like coat of bloody mucus covering the entire intestinal tract.

Acute gastric distention is a seldom encountered and not adequately explained postoperative phenomenon that is a serious emergency. The pain and interference with respiration may rapidly produce a shocklike state with rapid exitus of a patient in poor condition.

Decreased gastrointestinal tone and motility are present long after the patient has apparently reacted. These are usually recovered by forty-eight hours, but should they persist, edema and injection of the mucous membrane may be seen, and the bowel appears lax and dilated. This is the picture of paralytic ileus. The volatile anesthetics are the chief offenders, although it is encountered after all types of anesthesia—infection and surgical handling seeming to play the major part in its development.

Nausea and vomiting after an inhalation anesthetic are more common and more severe if the anesthesia has been stormy and the surgery difficult. These complications, by adding dehydration and electrolyte imbalance to the already depressed renal, hepatic, pancreatic, and intestinal functions may become self-perpetuating.

A severe proctitis sometimes follows the administration of a rectal anesthetic agent—ether and decomposed avertin being the chief offenders. Secondary infection in the gangrenous tissue may lead to persistent ulcerations, sloughs, and strictures.

Liver Pathology

The liver suffers primarily from halogen poisoning, although ether produces cloudy swelling of the parenchyma, and vinethene, if used for other than short procedures, can produce lesions like those of chloroform. Chloroform always damages the liver, but the damage is usually reversible. Fatal cases show the classical central lobular necrosis and fatty degeneration. An almost identical picture may be produced by long anesthetic procedures with vinethene. Trichlorethylene was recently involved in an acute fatty degenerative process showing central, midzone, and peripheral necrosis. Avertin, given in dosage of 80 milligrams/kilogram to an apparently normal little girl, is said to have caused death from acute fatty degeneration in a few days. These livers are pale, yellowed, soft, with swollen rounded edges, and the cut surfaces are greasy to touch. The cells, where not necrotic, are filled with droplets of fat.

Eclampsia, in contrast to the halogen compounds, presents a peripheral lobular necrosis, with the central areas remaining fairly intact.

Kidney Lesions

In the eclamptic kidney, there is a marked narrowing of all of the glomerular capillaries, due to the thickening of the capillary basement membrane. In rare cases, symmetrical necrosis of the renal cortex has been described as a terminal event—there being only a thin layer of normal tissue beneath the capsule (supplied by anastomoses from the capsular arteries).

Postoperatively, the volatile anesthetics produce cloudy swelling of the parenchyma of the kidney. Should there be present a glomerulonephritis—chronic or acute—an exacerbation may occur from the irritation of the cells, and further depression of function may be fatal. Avertin, conjugated with glycuronic acid in the liver, is excreted as the glycuronate by the kidney, with some reversible damage to the kidney occurring in the process. In the presence of renal disease, avertin and the other halogen compounds may cause an acute fatty degeneration of the tubules.

The kidney is the chief sufferer after a hemolytic transfusion reaction. After the prodromal symptoms, the appearance of icterus indicates the presence of an excessive amount of free hemoglobin. Hemoglobinuria followed by oliguria or anuria are the clinical evidences of the pathologic processes taking place in the kidneys. Examination at this time would show sharply demarcated cortical and medullary tissue, with a slightly reddish brown tinge to the medullary areas. The epithelium of the secretory tubules would be seen to be full of hematin crystals and granules. The larger tubules would be plugged with granular casts, and the collecting tubules would be seen to be full of hematin crystal masses. The stroma would be somewhat edematous. A variable degree of degeneration of the tubular epithelium would take place. Some of the patients die in uremia. In others a diuresis occurs and the condition gradually clears over a period of months. Several autopsies have shown, in addition to the renal lesions, focal areas of necrosis in the central zone or inner third of the liver lobules, with the Kupffer cells filled with black-brown pigment.

Central Nervous System Lesions

The pathogenesis and pathology of the central nervous system lesions suffered under general anesthesia have been discussed under the heading "Oxygen Want." These same complications may arise during spinal anesthesia. Here the oxygen deficiency is effected by intercostal or diaphragmatic paralysis, by severe hypotension with consequent cerebral anemia, or by mechanical interferences with an already partially paralyzed respiratory mechanism (large abdom-

inal packs, retractors at the costal margins, heavy assistants, and awkward operating positions).

The most frequent of these is the pounding headache which occurs postoperatively. Little is described histologically, since these patients do not die. Theories of causation include increase of spinal fluid pressure, decrease of spinal fluid pressure, the size of the spinal needle, the entrance of air into the subarachnoid space, the introduction of irritating chemicals along with the anesthetic agent, and a direct toxic effect of the agent upon the meninges or cord or brain substance. All of the hypotheses have been substantiated in part, but none explain even the majority of headaches.

The second most common complication is urinary retention, although the effects of the operation, the opiates commonly used postoperatively, and the postoperative supine position make this phenomenon difficult to evaluate as a purely anesthetic complication. Once the bladder has become distended, the overstretching of the muscles makes return of voluntary function difficult. Multiple submucosal and intramuscular hemorrhages make a fertile field for secondary infection, and the condition tends to persist unless treated promptly.

Cranial nerve palsies occupy the next position on the list, the abducens nerve being the usual victim. This is the nerve that also first loses its function in toxic encephalopathies, alcoholism, and increased intracranial pressure. Its vulnerability is said to be due to its small diameter and long course (although the trochlear nerve is more tenuous and has a longer course), its being fixed at the pons and cavernous sinus, its angle turn over the apex of the petrous portion of the temporal bone, and the crossing of it at right angles by the inferior cerebellar artery and the internal auditory artery.

All of the other cranial nerves except the olfactory, glossopharyngeal, and vagus have been involved in postspinal palsies, but pathologic data is lacking.

Rarer sequelae include the cauda equina syndrome, peripheral neuritis, septic and aseptic meningitis, epidural abscesses, arachnoiditis, toxic myelopathies, and perforation of the intervertebral discs by the spinal needle.

Davis and his associates have shown that the commonly used spinal anesthetic agents are both hemolytic and myelolytic. After Lundy reported that procaine in concentrations under 17.5 per cent produced no toxic changes, MacDonald claimed to be able regularly to produce destruction of nerve tissue in cats with 10 per cent concentrations. Davis demonstrated, in dogs and cats, varying degrees of inflammatory reaction in the leptomeninges, ganglion

cell changes of retrograde type similar to those seen in Wallerian degeneration, swelling and fragmentation of the axis cylinders, and degenerative changes in the fibers of the cord. Sections of human tissue suffering from clinical "burning" of the cord by the spinal anesthetic agent resemble the picture described by Davis. There is extreme destruction of the myelin sheaths, axis cylinders and glia, chiefly at the periphery of the cord and at the zones of entrance of the posterior roots. The anterior and lateral horn cells are also involved, in a manner faintly reminiscent of that seen in amyotrophic lateral sclerosis. Cords already involved with pernicious anemia, multiple sclerosis, tabes, and other neuropathies may show extension of these processes, and general paresis may be exacerbated.

The inflammatory reaction in the leptomeninges described by Davis in animals is represented in human material by varying degrees of arachnoiditis. This discouraging complication often makes its appearance after the patient has left the hospital, and may not be reported to the anesthetist or surgeon. The bizarre combinations of motor, sensory, and sympathetic symptoms are explained by the irregular adhesions of the meninges to the cord and nerve roots. These adhesions may be dense and massive or delicate and few. A woman was found to have the cauda equina and condus medullaris irreparably entangled after receiving 125 mg. of procaine for a hysterectomy. A man presented a complete block of the subarachnoid space from T-4 to T-10 after a segmental spinal for gastric resection with 2 per cent metycaine. The condition is chronic, progressive, and difficult to treat.

The frequency and severity of neurologic complications seem to bear a direct relationship to the concentration of the drug, and the nerves that are exposed to the greatest concentration of the drug as it leaves the spinal needle are the ones that suffer the most. Eversole states that the concentrations commonly employed in anesthesia fall little short of those which would cause neurologic complications in a much greater percentage of patients. It would be wise to test for spinal block before administering any spinal anesthetic, but Poppen and Hurxthal claim that the cerebrospinal fluid dynamics remain apparently normal until the opening of the subarachnoid space is smaller than that of the spinal needle.

The position of the peripheral nerve palsies as isolated neurologic complications is precarious until it can be determined that direct trauma was not at fault. The two nerves most commonly involved—the ulnar and the peroneal—are the two most likely to be injured on the operating table.

As the needle pierces the skin and enters the subarachnoid space, it may carry noxious material which can be injected with the anesthetic agent. Several cases of frankly septic meningitis have been described, and many cases of the so-called "chemical meningitis" have been reported. The latter was found by Orkin, in a review of over 46,000 spinal anesthetics, to occur about once in each 250 cases. Chemical contamination of the anesthetic solution has usually been blamed. In the septic type, the sugar is low and the organisms may be demonstrated. In the "aseptic" type, the sugar is normal and the protein high. The pressure cell count is elevated in both types.

A brief discussion of the action of local anesthetics in oil should be included in this talk. Studies have shown that the action of these substances, if prolonged, is due to destruction of the nerve fibers rather than prolonged contact made possible by the vehicle. The more fibers destroyed, the longer the action. Recovery in the experimental animal was complete, although pooling of the oil with abscess formation might theoretically prevent reformation of the nervous pathway.

Miscellaneous Conditions

Convulsions while under general anesthesia may be due to many causes—oxygen deficiency, carbon dioxide excess, cocaine derivatives, surgical stimulation of brain tissue, and epilepsy. The usual so-called "ether convulsion" is typically encountered in a septic, dehydrated, febrile patient, usually young, after prolonged deep anesthesia in an operating position where respiratory exchange is disturbed. It has been postulated that respiratory acidosis and poor oxygenation, added to a preexisting tissue acidosis gives rise to these convulsions. The only constant changes at autopsy have been capillary congestion and petechial hemorrhages of the brain, heart, kidneys, and serous membranes, all consistent with the changes seen in acute oxygen want.

Status thymolymphaticus is a traditional diagnosis used as an explanation for sudden exitus on the operating table. While it is true that some patients do not tolerate general anesthesia as well as others, endocrine dysfunction has not been shown to be a factor except in frank cases of hypothyroidism or Addison's disease. A rare patient, otherwise normal, requires extremely minute amounts of general anesthetic agents. This type of person, usually scrawny and asthenic, may drop to respiratory arrest after only a few breaths of a powerful anesthetic agent. Should this agent be ethyl chloride or chloroform, cardiac arrest may occur simultaneously with respiratory arrest. During the resultant con-

fusion, a cubic centimeter of epinephrine injected into the heart usually finishes the patient. The presence of a gas machine for artificial respiration with oxygen will save most of these patients, sometimes even in spite of the intracardiac epinephrine that is invariably given.

The vagovagal reflex is a more modern and more fashionable explanation for some cases of sudden death on the operating table. This highly controversial reflex is elicited by stimulation of the larynx or trachea, and manifested by abrupt circulatory and respiratory arrest. Some authors maintain that concomitant hypoxia is necessary for the elicitation of the reflex—the hypoxia sensitizing the myocardium.

Carbon Dioxide and Local Anesthetics

Deaths due to excess of carbon dioxide and those due to the actions of local anesthetic agents bear a certain similarity. In each case there is at first central nervous system stimulation and then depression, with convulsions and coma as terminal events. Both carbon dioxide and local anesthetic agents can seriously affect the conducting mechanism of the heart. The local anesthetic agents may, in large concentrations, affect the heart muscle directly, with terminal ventricular fibrillation and final cardiac standstill in the diastolic phase.

The pathologic findings are disappointing, showing only those features consistent with acute oxygen want. Death is apparently due to circulatory failure, or central respiratory depression.

Summary

An attempt has been made to discuss, briefly, some of the pathologic pictures and processes peculiar to anesthetic agents and technics. As surgical procedures increase in length and complexity, and as the poorer risk patients are now considered suitable for many major surgical procedures, anesthesia has of necessity become more of a problem, and anesthetic complications should be borne in mind during the preoperative study of these patients, and should be watched for during the operative and postoperative course.

References

Adriani, J.: The Pharmacology of Anesthetic Drugs, 2nd ed. 4th printing, Charles C. Thomas, Publisher, Springfield, Illinois.

Adriani, J.: Some Hazards of Anesthesia, New Orleans M. & S. J. *98:*324 (Jan.) 1946.

Andersen, D. H.: Avertin Poisoning with Acute Yellow Atrophy of the Liver and Toxic Nephrosis, Anesthesiology *6:*284-301 (May) 1945.

Barach, A. L., and Rovenstine, E. A.: Hazard of Anoxia during Nitrous Oxide Anesthesia, Anesthesiology *6:*449-461 (Sept.) 1945.

Barbour, J. H., and Seevers, M. H.: A Comparison of the Acute and Chronic Toxicity of Carbon Dioxide with Especial Reference to its Narcotic Activity, J. Pharmacol. & Exper. Therap. *78:*11 (May) 1943.

Beecher, H. K.: The Physiology of Anesthesia. New York: Oxford University Press, 1938.

Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, 4th ed., The Williams & Wilkins Co., Baltimore.

Black, M. G.: Spinal Fluid Findings in Spinal Anesthesia, Anesthesiology *8:*382 (July) 1947.

Bourne, W.: Anesthetics and Liver Function, Am. J. Surg. *34:*486, 1936.

Boyd, W.: A Textbook of Pathology, 3rd ed., Lea & Febiger, Philadelphia.

Brocks, B. A., and Davison, C.: Nervous Complications following Spinal Anesthesia; A Clinical Study of Seven Cases with Tissue Study in One Case, J.A.M.A. *106:*441 (Feb. 8) 1936.

Brown, E. W.: The Physiologic Effects of High Concentrations of Carbon Dioxide, U. S. Nav. M. Bull. *28:*721 (Oct.) 1930.

Brown, R.: Osteomyelitis of the Spine Secondary to Paravertebral Block, U. S. Army M. Bull. *8:*391-396 (May) 1948.

Burton, L. W.: Granuloma of the Larynx; A Late Complication of Endotracheal Anesthesia, Ann. Otol., Rhin. & Laryng. *56:*191-193 (March) 1947.

Cole, F.: Explosions in Anesthesia; Review of Literature, Surgery *18:*7-26 (July) 1945.

Cole, F.: A New Lethal Dose of Curare, with Some Observations on the Pathology produced by Large Doses, Anesthesiology *7:*190-197 (March) 1946.

Coleman, F. P.: Effects of Anesthesia on Hepatic Function, Surgery *3:*87 (Jan.) 1938.

Courville, C. B.: Untoward Effects of Nitrous Oxide Anesthesia with Particular Reference to Neurologic and Psychiatric Manifestations. Mountain View, California: Pacific Press Pub. Assoc., 1939.

Courville, C. B.: Asphyxia as a Consequence of Nitrous Oxide Anesthesia, Medicine *15:*199-242, 1936.

Courville, C. B.: Ether Anesthesia and Cerebral Anoxia; A Study of the Causative Factors, Anesthesiology *2:*44 (Jan) 1949.

Davis, L.; Haven, H.; Givens, J. H., and Emmett, J.: Effects of Spinal Anesthesia on the Spinal Cord and its Membranes, J.A.M.A. *97:*1781 (Dec.) 1931.

Diddle, A. W., and Hill, A. M.: Pulmonary Embolism during Continuous Caudal Analgesia, West. J. Surg. *51:*427, 1943.

Donnelly, W. A.; Grossman, A. A., and Grem, F. M.: Local Sequelae of Endotracheal Anesthesia, Anesthesiology *9:*490 (Sept.) 1948.

Gauss, H.: The Pathology of Fat Embolism, Internat. Clin., 23rd Series, *4:*171-227, 1913.

Gready, T. G.: Some Complications of Caudal Anesthesia and their Management, J.A.M.A. *123:*671, 1943.

Harroun, R.; Beckert, F. E., and Fisher, C. W.: The Physiologic Effects of Curare and its Use as an Adjunct to Anesthesia, Surg., Gynec. & Obst. *84:*491 (April) 1947.

Herbert, P. A.: Renal Changes in Secondary Shock, Ann. Int. Med. *25:*648 (Oct.) 1946.

Hingson, R. A., and Edwards, W. B.: An Analysis of the First Ten Thousand Confinements Managed with Continuous Caudal Analgesia, with a Report of the Authors' First Thousand Cases, J.A.M.A. *123:*538, 1943.

Holmes, R.: An Analysis of Pulmonary Complications following Anesthesia in 2,064 Consecutive Cases, Anaesthesia *3:*67 (April) 1948.

Kennedy, F.; Somberg, H., and Goldberg, B. R.: Arachnoiditis and Paralysis following Spinal Anesthesia, J.A.M.A. *129:*664, 1945.

Landmesser, C. M.: A Study of the Bronchoconstrictor and Hypotensive Actions of Curarizing Drugs, Anesthesiology *8:*506 (Sept.) 1947.

Lichenstein, L., and Sewall, S.: Fat Embolism, J.A.M.A. *136:*827 (March 20) 1948.

Light, G.; Sweet, W. H., and Livingstone, H M.: Neurologic Changes following Spinal Anesthesia, Surgery *7:*138-156 (Jan.) 1940.

Livingstone, H.; Wellman, V.; Clark, D., and Lambros, V.: So-called "Aseptic or Chemical Meningitis," Surg., Gynec. & Obst. *77:*216 (July) 1943.

Long, J. H.; Oppenheimer, M. J.; Wester, M. R., and Durant, T. M.: The Effect of Intravenous Procaine on the Heart, Anesthesiology *10:*406 (July) 1949.

Loring, A. L.; Jacoby, J. J., and Livingstone, H. M.: Studies on the Parenteral Administration of Hydrogen Peroxide, Anesthesiology *9:*162 (March) 1948.

Lundy, J. S.; Essex, H. E., and Kernohan, J. W.: Experiments with Anesthetics: IV: Lesions Produced in the Spinal Cords of Dogs by a Dose of Procaine Hydrochloride Sufficient to cause Permanent and Fatal Paralysis, J.A.M.A. *101:*1546 (Nov. 11) 1933.

MacDonald, A. D., and Watkins, K. H.: An Experimental Investigation into the Cause of Paralysis following Spinal Anesthesia, Brit. J. Surg. *25:*879, 1938.

Macklin, C. C.: Pneumothorax with Massive Collapse from Experimental Local Overinflation of the Lung Substance, Canad. M. A. J. *36:*414 (April) 1937.

Meek, W. J.; Hathaway, H. R., and Orth, O. S.: The Effects of Ether, Chloroform and Cyclopropane on Cardiac Automaticity, J. Pharmacol. & Exper. Therap. *61:*240 (Nov.) 1937.

Mousel, L. H.; Stubbs, D., and Kreiselman, J.: Anesthetic Complications and their Management, Anesthesiology *7:*69-79 (Jan.) 1946.

Nicholson, M. J.: Hemolytic Transfusion Reactions, Anesthesiology *9:*345 (July) 1948.

Orkin, L. D.: Reported Mortality and Morbidity following Spinal Anesthesia, American Society of Regional Anesthesia, Scientific Session, March 3, 1936.

Parke, W. M.; Loftus, E. R., and Bishop, H. F.: The Incidence of Pulmonary Tubercular "Spreads" following Thoracic Surgery and Anesthesia, New York State J. Med. *48:*1685 (Aug.) 1948.

Pitkin, G. P.: Conduction Anesthesia. Philadelphia: The J. B. Lippincott Co., 1946.

Pisetsky, J. E.: Hemiplegia Following Ether Anesthesia, Anesthesiology *6:*522-527 (Sept.) 1945.

Pott, H. M.; Tobias, J. M.; Swift, M. N.; Postel, S., and Gerard, R. W.: Hemodynamics in Pulmonary Irritant Poisoning, Am. J. Physiol. *147:*329 (Oct.) 1946.

Rosenthal, S. M., and Bourne, W.: The Effect of Anesthetics on Liver Function, J.A.M.A. *90:*377, 1928.

Ruth, H. S.: Anesthesia Study Commission, Findings of Eleven Years, Philadelphia Med. *43:*1157 (March 27) 1948.

Sanders, J., and Isoe, I.: Intravenous Oxygen and Pulmonary Embolism, Ann. Surg. *126:*208 (Aug.) 1947.

Schnedort, J. C.; Johran, P. H., and Orr, T. G.: The Problem of Anoxia in Surgery and Anesthesia: Report of Experimental and Clinical Cases and a Review of the Literature, Arch. Surg. *43:*169 (Aug.) 1941.

Slocum, H. C.; Hoeflich, E. A., and Allen, C. R.: Circulatory and Respiratory Distress from Extreme Positions on the Operating Table, Surg., Gynec. & Obst. *84:*1051 (June) 1947.

Strumia, M. H.: Post-transfusion Hemalytic Reactions, Pennsylvania M. J. *47:*668 (April) 1944.

Thorner, M. W., and Levy, F. H.: The Effects of Repeated Anoxia on the Brain, J.A.M.A. *115:*1595, 1940.

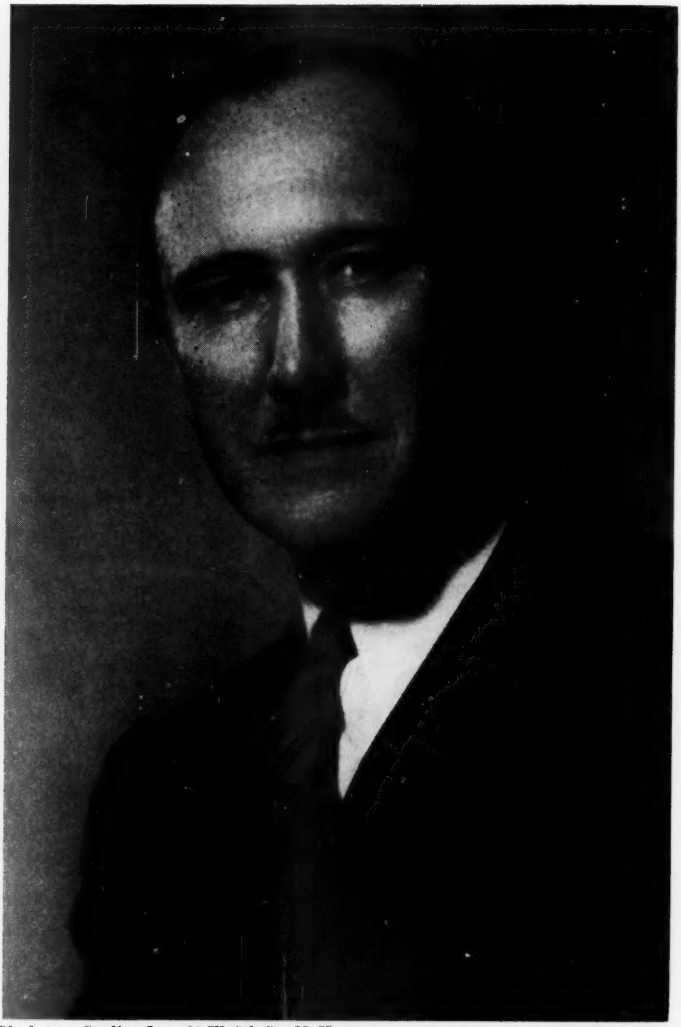
Valle, A. R.: Lung Abscess, An Analysis of 244 cases, Surg., Gynec. & Obst. *21:*278 (Sept.) 1945.

Van Allen, C. M.; Hardina, L. S., and Clark, J.: Air Embolism from the Pulmonary Vein; A Clinical and Experimental Study, Arch. Surg. *19:*567 (Oct.) 1929.

Waters, R. M., and Gillespie, N. A.: Deaths in the Operating Room, Anesthesiology *5:*113 (March) 1944.

Waters, R. M.: Toxic Effects of Carbon Dioxide, New Orleans M. & S. J. *90:*219 (Oct.) 1937.

Zweitel, E.: Fatalities after Sacral Anesthesia, Abstracted in J.A.M.A. *74:*1138, 1930.



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THE UNIVERSITY OF LOUISVILLE

The University of Louisville School of Medicine is very proud that it has been selected to sponsor this issue of THE SOUTHERN SURGEON. A School which has provided 9 surgeons as Presidents of the American Medical Association, which has furnished the first American President (-elect) of the World Medical Association, which has supplied many first-officers to numerous surgical societies, and with which are associated such famous names-in-surgery as Daniel Drake, Samuel Gross, Russell Hibbs, D. W. Yandell, Louis McMurtry and Irvin Abell (to name a few), has a tradition behind it which should serve as a powerful incentive to those men who are carrying on the work of their illustrious predecessors. We hope that the reports appearing here will, in their caliber and quality, be regarded as consistent with the standards set by this tradition.

The surgeons of yesteryear had few laboratories in which to work, little in the way of expensive equipment apart from their surgical instruments, and none of the expansive and expensive Research Institutes of today in which to work. They did have some animals available for developing and perfecting new technics—and they had their patients to work with. All too frequently, their patients had to serve as experimental animals, for there was no other way to study new methods and technics save that of trial and

error. Sometimes they had brilliant successes immediately, but sometimes—as in the case of Marion Sims' efforts to repair vesicovaginal fistula—they achieved success only after many discouraging failures, in which it was the patient, after all, who was the real victim of the failures. But those pioneers did have the personal qualities of imagination, of energy, of ingenuity and of sheer nerve, which made up, to some extent, for lack of the facilities of which we boast today.

That is not to infer that those personal qualities are lacking in the surgeon of today, for they are probably just as generally distributed now as then. But it is no longer necessary for the surgeon to take chances with human lives in his quest for new knowledge. Today the laboratory is just as important to the inquisitive surgeon as to the internist; today animal experimentation precedes human application of new technics, as in the instance of the Taussig-Blalock operations for congenital heart disease; today the radiologist, the bacteriologist, the pathologist and the biochemist are working in closer cooperation with the surgeon, in both diagnostic and therapeutic procedures. In the larger research centers, the surgical departments are involved in extensive laboratory and animal experimentation studying a great variety of problems, many of which superficially appear to be of more immediate concern to the internist than to the surgeon. Thus does the gulf between surgery and the nonsurgical branches of medicine become narrowed—which is as it should be.

As would be anticipated, scientific reports emanating from centers where extensive laboratory and animal experimentation is possible are apt to deal with fundamental and basic problems. Conversely, surgical reports from clinics where such facilities are meager or absent must necessarily be of an applied or clinical or statistical nature; investigations of this kind are valuable, sometimes fundamentally so, and it is on that basis that the fame of most of the great men of surgery was earned. But today, the surgeon with the inquiring mind wants to pry into basic and fundamental phenomena, and to develop technics without jeopardizing the life or health of human beings in the process. For this purpose, he needs the laboratories and the facilities aforementioned. Now, a few medical schools are in the fortunate position of being financially able to provide such facilities from their own budgets. Unfortunately, most schools are not so well situated. For these, it is usually possible to procure help from the Federal Government or other organizations in the form of grants-in-aid. But, except in certain instances, these grants do not provide for construction, though they will pay for staff, equipment, maintenance, et cetera. Since the very schools which

need this aid most urgently are usually unable to furnish funds from their own budgets for construction of facilities, and since, very often, they cannot use prospective aid effectively because there is no place for the staff to work, a "vicious circle" is thereby created, which can be broken only by the creation of working-space. It would seem that the Public Health Service and other agencies would vastly increase their potentialities for increasing productive research, if they were to liberalize their policies regarding grants for construction, so that the very schools which need the help most badly could develop active programs in basic investigation, and thereby dispel the sense of frustration which the "vicious circle" in which they are caught constantly produces. In other words, if the present situation of "them that has, gets" could be changed to "them that hasn't, may also get," the research possibilities in the medical schools of the entire country would be tremendously stimulated. One recognizes the possibilities of abuse, but it is common knowledge that abuses exist even under the present policies, and it is doubtful that the percentage of abuse would be increased by such liberalization of policy. In fact, if deserving schools and institutions knew that it would be possible to obtain Construction Grants for badly needed facilities, it is probable that they would search their consciences very carefully before making requests for such aid.

Those schools which are blessed with ample facilities for surgical research may be expected, therefore, to produce reports dealing, to a very large extent, with investigations into fundamental principles. Those schools whose research facilities are meager and whose budgets are strained are not prevented thereby from research activities, but these are perforce pretty much restricted to the clinical field; nevertheless, this field is so unlimited in its scope, and the possibilities are so boundless, that it is still possible to produce results of the first magnitude, especially in the field of surgical technics. In between these two extremes lie those schools—and they probably represent the bulk of the medical schools in the country—which do possess some facilities for fundamental research and a competent staff to carry it on, but which have the means to do a great deal more of it, if only they had a suitable place in which to work and in which to keep their experimental animals.

The University of Louisville is in the latter class. Facilities for basic research in surgery do exist here, but they are considerably limited. Consequently, the papers in this number of THE SOUTHERN SURGEON are primarily of a clinical nature and do not announce any startling new discoveries. But we hope that these reports of the experience of the members of our surgical department will be a valuable addition to the sum total of knowledge in this department of

medical science. Some of these papers do represent original and fundamental investigation and we hope that they will be worthy of publication.

As an internist, this writer believes that when one branch of medicine advances and gathers new information, all of medicine gains thereby. Those of us who are not surgeons are therefore happy that the surgical service of our School has this opportunity to "show its stuff," and we hope that you, the reader, will find it good.

J. MURRAY KINSMAN, M.D.
Dean

CHOLELITHIASIS—A SURGICAL DISEASE*

Improvements in roentgen technic, in addition to more frequent and more thorough exploration at laparotomies, have resulted in an increasing number of diagnoses of cholelithiasis. Many of these cases are symptomatic, others are not. When one understands the pathologic processes and distressing symptoms gallstones can cause, it is surprising that there is still a difference of opinion regarding this treatment.

None of us believe that stones can be dissolved or extruded by medication, yet such cases are still treated by medicine and diet and colics relieved by sedation in many instances.

Pain is a great persuader. Gallstone colic, painful digestive distress, reflex spasm of the pylorus and gaseous distention with pressure symptoms in the chest will often persuade the patient to seek surgical relief. Not many physicians or patients will question this choice of treatment.

It is the so-called silent stones that cause controversy regarding their proper management.

A careful history will often show that such are not silent at all. Even in the cases where this is true, one must always bear in mind that silent stones are not harmless stones.

The significance of both stones causing symptoms and those that do not is dangerously difficult to evaluate. This is because there is so often a marked inconsistency between the prominence of the symptoms and the degree of pathologic changes present—much greater than is seen, for instance, in appendicitis.

The surgical mortality and morbidity, in uncomplicated cases of cholelithiasis, are both low and the preoperative and postoperative care is now so effective that even the aged and those suffering with various chronic diseases can be made relatively safe risks. When one contrasts the usual smooth surgical course in the simple cases with the stormy one when complications are present, he should be easily convinced of the wisdom of avoiding such complications.

These sequences of simple cholelithiasis are a roll call of some of the most dangerous surgical lesions of the upper abdomen—and rarely are such conditions found in the absence of gallstones. Some of the commoner ones should be mentioned.

Acute cholelithiasis with gangrene, perforation and peritonitis is practically always obstructive in nature and usually the obstruction is due to stones.

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Cholangitis and common direct obstruction are nearly always secondary, in time at least, to long-standing stones in the gallbladder.

Fistulae between the gallbladder and the intestinal tract are usually due to stone necrosis.

Even intestinal obstruction may develop if the fistula permits the passage of a sufficiently large stone.

Acute pancreatitis is so often associated with gallstones that many believe that the calculus obstruction at the opening of the pancreatic ducts is the prime etiologic factor in this most serious disease. Chronic pancreatitis may arise from various causes but usually is a part of the chain of biliary tract disease.

Carcinoma of the biliary tract on the head of the pancreas is another condition found only too often with an associated cholelithiasis. The surgical mortality in uncomplicated cases of simple gallstones compares very favorably with the incidence of incurable cancer in that area.

In long standing cases of gallstone disease there is often an associated hepatitis. When it occurs postoperative relief may not be entirely satisfactory.

There are several types of jaundice but the obstructive variety is generally due to stones or their sequellae. Even in non-fatal cases the effects of jaundice may be most disabling.

In summary it would, therefore, seem fair to state that the removal of silent or asymptomatic gallstones is a safe procedure surgically; that good health is compatible with cholecystectomy which is the procedure of choice to avoid further stone formation; and that most of the serious surgical diseases of the right upper quadrant will be avoided if such early removal is accomplished.

General adherence to this policy over a period of years would contribute definitely to increased longevity.

J. DUFFY HANCOCK, M.D.